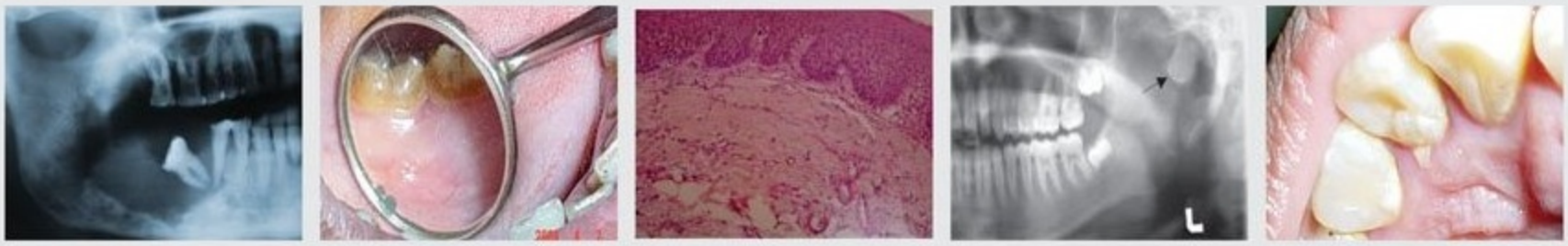

Fundamentals of Oral Medicine & Radiology

Editors
Durgesh N Bailoor
KS Nagesh

JAYPEE

Fundamentals of Oral Medicine and Radiology
Bailoor
Nagesh

VISIT…

Fundamentals of Oral Medicine and Radiology

DISCLAIMER NOTICE

This book is a supplement and not a replacement for professional dental training. The information in this text should not be used by unqualified personnel to do any self-diagnosis. All dental surgeons are requested to kindly verify the latest prescribing practices with your teachers and consultants prior to making real life decisions. Most values are indicative and have been checked against latest reliable sources, but the publishers and editors do not have any direct or indirect liability to the use or misuse of this prescribing information.

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Fundamentals of Oral Medicine and Radiology

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Fundamentals of Oral Medicine and Radiology

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We dedicate this book to our teachers who made us what we are and our students who taught us so much…….

Durgesh N Bailoor and KS Nagesh

Learning is finding out what you already know,
Doing is demonstrating that you know it,
Teaching is reminding others that they know
Just as well as you……
……***You are all*** *Learners, Doers and Tea*chers

Richard Bach. 1989
"Illusions"
The adventures of the
reluctant messiah

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Our families have borne the neglect and moodiness which goes with doing any work of this magnitude, to them we are eternally grateful. The staff of department of Oral medicine of RV Dental College, Bangalore and of Department of Yenepoya Dental College and Hospital, Mangalore are both saluted for their contribution and help.

The postgraduates of the Yenepoya Dental College and Hospital, Dept of Oral Medicine and Radiology Prasanna Kumar, Nillofer Shabnam, Beena Kumari, Omal PM, Ajay Nayak, Kiran K, Sham Kishore and Phillips Mathew. All have contributed their time and energies in proofreading and cross verifying references. Our thanks to them for their dedication.

Our thanks to Prasanna Kumar who has contributed to various line diagrams and Prof Akhter Husain and Yasser who have helped creatively in the cover design.

We have learned at the feet of our venerable teachers , we have understood things better because our BDS and MDS students enlightened us with their discussions and queries. Many ideas that are claimed to be ours are really the vision that we saw by standing on the shoulders of the giants of oral medicine and radiology.

The mistakes that will inevitably creep in are our responsibility alone; please point them out to us, so we can improve the next edition.

Dr. B. H. SRIPATHI RAO, M.D.S.

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Foreword

It gives me great pleasure to write a foreword to this book

Fundamentals of Oral Medicine and Radiology, 3rd edition' edited by two senior professors Dr Durgesh Bailoor and Dr K S Nagesh in the field of Oral Medicine and Radiology.

This book is the first multi-authored textbook in the subject of Oral Medicine and Radiology published by Indian authors. A total of thirty-eight professionals from multidisciplinary areas have contributed and done peer review. A lot of Indian statistics and references makes this a relevant text for students of all categories and the practicing dentist. Flow charts, diagrams and clinical pictures enhance the teaching potential of this book.

Editors of this text are one of the first to introduce concepts in oral psychosomatic medicine, computers in oral diagnosis and the use of complementary and alternative medicine systems in this field. The textbook also emphasises the need to understand principles and role of radiotherapy in management of oral cancer.

The emerging areas of CT, MRI AND SPECT scan are slowly moving from expensive to commonplace investigations. The digital revolution and the worldwide web have made knowledge dissemination instantaneous and accurate and its importance is highlighted to the student of this subject.

We are seeing a plethora of Indian authors bringing out learned tomes for our next generation to follow. This healthy trend is catalyzed by Indian publishers like M/s Jaypee Brothers Medical Publishers (P) Ltd, who are bringing the innovative technologies in printing and publishing to the students and doctors of health care.

This book is an excellent contribution to our scientific literature in Indian scenario thereby facilitating our students to understand the diseases pattern that exist in developing country like ours.

Prof BH Sripathi Rao
Principal, Yenepoya Dental College, Mangalore
Executive Member of Dental Council of India, New Delhi

Preface

It is with a great sense of satisfaction that we present this edition to the new generation of dental students and practitioners. We have updated all the references to the latest possible and tried to present a median view wherever two schools of thought have clashed.

As far as possible, the relevance of dental medicine knowledge, as required by the dentists of the developing world is kept in mind. Indian research and Epidemiology has been quoted where available.

The concepts of oral psychosomatic medicine, computers in dental practice, radiotherapy for oral cancer and alternative therapies in the oral diseases have been presented in this book for the first time. It is with great pride we state that this book is in its third edition, now with Jaypee Brothers Medical Publishers (P) Ltd. First was in 1994 and the second edition in 2001. It remains till date the first multi-authored peer reviewed book for practicing doctors ever published in India in this specialty.

Our contributing authors have ranged from oldies, like principals and vice-principals to young turks like the recently passed postgraduates with new and bubbling ideas of the cyber and robotics age. This healthy mix we feel will nurture the growing dental mind better.

We salute our teachers for guiding us and thank the students for being catalysts in our quest for wisdom.

We thank our families for putting up with our temper tantrums during the arduous journey in production of this manuscript.

Durgesh N Bailoor
KS Nagesh

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1 The Diagnostic Sequence

"Learn to see, learn to hear, learn to feel, learn to smell and know that by practice alone you can become an expert."
—Sir William Osler

DEFINITION

History

History is defined as planned professional conversation followed by accurate recording of facts. Symptoms are primarily subjective complaints told or expressed by the patient who, may or may not, have externally observable element, e.g. Angina pectoris, neuralgic pain etc. Signs are those clinical entities, which the doctor can observe and record as objective findings, e.g. Pallor, Bulla, etc.

History is classified as two types – structured and unstructured.

- *Structured history* consists of pre-decided format or a printed form in which questions can be asked in logical manner. In recent time computers have been programmed for structured history taking. Bertoft G[6] (1996) in his retrospective study mentions how structured medical and dental history helps in diagnosis of oro-facial pain, TMD symptoms and evaluation of various psychological factors and is a strong proponent of this type of history.
- *Unstructured history taking*. Clinicians with experience or senior consultants frequently appear to ask unrelated question and come to a fairly accurate diagnosis; they change the pattern of questions as per the patient's narration. They are casual but penetrating and in perceptive way they may arrive at a diagnosis. This may seem magical to an uninitiated young doctor. It is actually years of discipline, reading and knowledge that go into this magic.

It is also now possible to look at Manual and Computerized type of record keeping. Most clinics and hospitals today have electronic record keeping of differing sophistication.

Diagnostic Sequence

This is series of steps that clinicians take to arrive at a diagnosis. Diagnosis is defined as the recognition of the disease, naming the disease as per agreed criteria. In other words, diagnosis would mean recognizing the disease and naming it.

ICD-DA or International classification of diseases to Dentistry and Stomatology[7] (1995) is a manual which gives a working clinician some kind of a codification which can help in noting the diagnosis as a number or using diagnostic words which are globally accepted. In research the use of ICD-DA numbers has proved invaluable for international communication and research (Fig. 1.2).

FIGURE 1.1: Listening carefully, recording meticulously and storing data systematically forms the cornerstone of good dental record making (Bailoor DN, Chatra LK 2004)

The Sequence

- Discovery either by patient or doctor of something abnormal * History taking * Clinical Examination * General * Extra Oral * Intra Oral * Clinical Diagnosis * Provisional Diagnosis * Investigations e.g. Hematology, Urine Analysis * Differential Diagnosis * Further Investigations (special tests) * Final Diagnosis * Treatment (Fig. 1.1).

When a set of closely appearing lesions are diagnosed then their enumeration and subsequent distinction from each other constitutes the differential diagnosis.

HISTORY AND COMPONENTS OF HISTORY[1-3]

History starts with recording the name, age, sex, marital status, occupation and address, which are collectively, called as identifying data. Next is the presenting complaint, or the chief complaint, the primary reason why the patient seeks the dentist's opinion. This complaint is recorded in patient's own words and further details are asked in the format of origin, duration, progress, and radiation. The aggravating and relieving factors are recorded. The impact of these symptoms on home and occupational life is also assessed.

Origin: Records how the problems started.

Duration: The temporal quantification, meaning how many days, weeks, or months, the problem has existed.

Progress: Denotes whether the problem is static, getting worse or getting better.

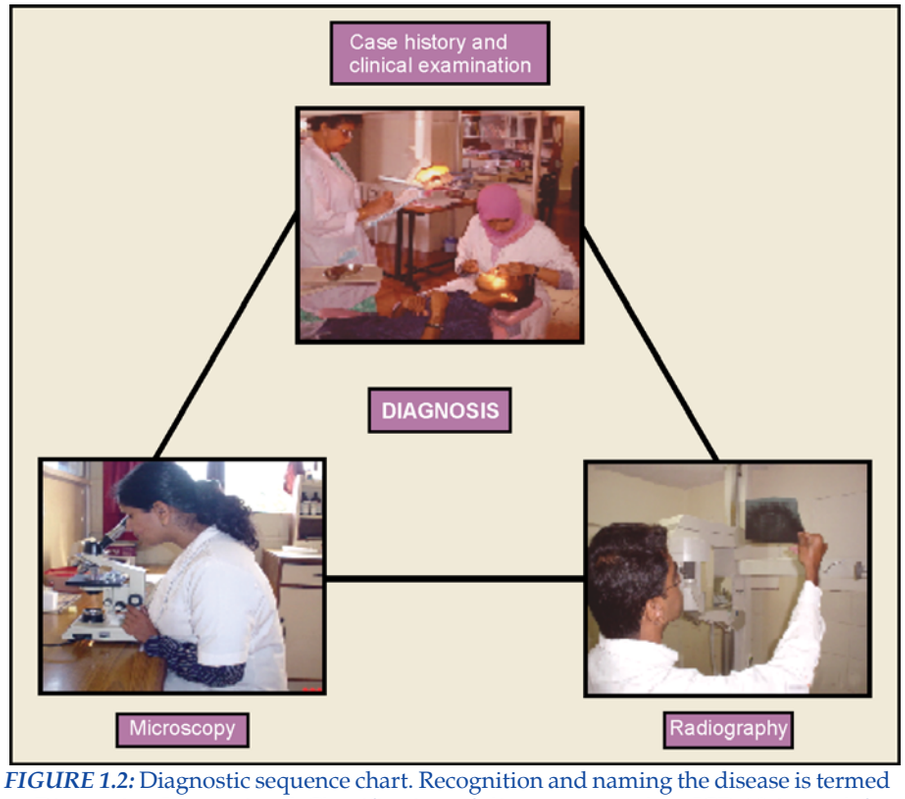


FIGURE 1.2: Diagnostic sequence chart. Recognition and naming the disease is termed as Diagnosis—International Classification of diseases termed as ICD-10 is used for global standardization (Beena K, Nillofer S, Omal P, Bailoor DN 2004. Yenepoya Dental College and Hospital, Mangalore, India)

Radiation: Indicates whether the problem is changing from one anatomic location to another, and also if it is changing in quality.

Past Dental History

This tells us whether the patient has been to a dentist before, what sort of treatment was done, what were the complications encountered. This part highlights the patient's attitude towards the dental treatment. Allergy to dental ointments, pastes mouth washes may also be recorded here.

1. Are you seeing a dentist regularly? Yes No
2. Do you bleed excessively after extraction? Yes No
3. Did you ever put braces? Yes No
4. Are you allergic to any injection, medicine or ointment applied to mouth? Yes No
5. Any other treatment. Yes No

Past Medical History

This can be recorded briefly by asking the following questions.

1. Are you seeing a family doctor for any illness now? Yes/No
2. Are you taking medications for any health problems? Yes/No
3. Are you allergic to any drugs, medicines, and food ? Yes/No
4. Were you hospitalized during the last five years for any major illness, operation, etc? Yes/No

If any of the questions is answered 'Yes', then a detailed questionnaire should be assessed. Such type of questionnaire has been termed by deJong KJ[5] (1997) as Medical risk-related history (MRRH). In his opinion the MRRH and personal interview follow up by the dentist,

would lead to accuracy in detection of medical problems of the dental patients.

Cardiovascular System

1. Do you have breathlessness on exertion like climbing stairs, walking fast, etc. Yes No
2. Do you have pain on the left side of the chest on exertion or emotional outburst? Yes No
3. Did you have any operation of the Chest, heart-valves etc in childhood? Yes No
4. Do you get spontaneous dizziness, palpitation with profuse sweating? Yes No
5. Did you ever get a stroke Yes No
6. Did you get sore throat, fever and fleeting joint pains in recent past? Yes No
7. Any other complaints. Yes No

Respiratory System

8. Do you have problems of wheezing? Yes No
9. Did you suffer from tuberculosis? Yes No
10. Did you have any sort of breathing problem in recent times? Yes No
11. Did you get swelling of ankles of legs? Yes No
12. Any other (Specify) Yes No

Gastrointestinal and Hepatic

13. Do you have heart burn/acidity? Yes No
14. Have you suffered from jaundice? Yes No
15. Bouts of nausea, lack of appetite? Yes No
16. Piles? Yes No
17. Persistent loose motions. Yes No

Endocrinal System

18. Do you have excessive thirst, hunger? Yes No
19. Do you have to urinate at night disturbing your sleep? Yes No
20. Do you feel that you have developed black patches on the skin, in mouth? Yes No
21. Have you gained or lost weight excessively in last three months? Yes No
22. Do you feel lethargic and drowsy recently? Yes No

Genitourinary

23. Do you get puffiness of the face? Yes No
24. Did you suffer from burning micturation? Yes No
25. Bouts of severe pain in lower back? Yes No
26. Any other. Yes No

Neurological

27. Do you get persistent headaches? Yes No
28. Do you have weakness of any one side? Yes No
29. Do you get blackout, loss of memory? Yes No
30. Have you had numbness, or tingling of fingers of hand and legs? Yes No
31. Any other. Yes No

Trauma

32. Did you meet with any major accident in recent times? Yes No
33. Any sports injury to facial region. Yes No
34. Any other. Yes No

Bleeding Disorders

35. Do you bleed easily on cutting yourself? Yes No
36. Are you taking any medication, which any make you bleed more (Anticoagulants?) Yes No
37. Do you bruise easily, get pin-point bleeding spots on skin or mouth? Yes No
38. Any other. Yes No
38. For women only:
    a. Are your menses regular? Yes No
    b. Are you pregnant? Yes No
    c. Any operations such as uterus removal, family planning, etc. Yes No
    d. Any other. Yes No

For both Men and Women

40. Were you treated for venereal disease? Yes No
41. Have you had any contact with a prostitute or sex worker? Yes No
42. Did you have more than one sex partner in last two years? Yes No
43. History of homosexuality? Yes No
44. Which countries did you travel recently, mention Yes No

45. Did you have blood transfusion recently? Yes No
46. Any other Yes No

Cranial Nerve Function

Note: If any of the questions is answered Yes the clinician must do a detailed clinical examination of the various functions of that cranial nerve. If serious deficit is detected or suspected, Neurologist's opinion is mandatory for a complete assessment.

47. Can you smell normally? CNI Yes No
48. Did you have any vision problems? CN2 Yes No
49. Are you able to move your eyeballs comfortably? CN3,4,6 Yes No
50. Are you able chew food normally, and feel the forehead? CN5 Yes No
51. Are you able to blow air into a balloon without difficulty? CN7 Yes No
52. Is your taste diminished or changed? CN9,CN10 Yes No
53. Do you feel that swallowing is a problem recently?CN9,CN10 Yes No
54. Do you feel increasing dryness of eyes? CN7 Yes No
55. Does your mouth run dry, recently? CN7,CN9 Yes No
56. Are you able to hear properly and maintain balance? CN8 Yes No
57. Has your ability to talk changed recently? CN 10 Yes No
58. Can you turn your head, and lift your shoulders? CN11 Yes No
59. Are you able to move your tongue just like before? CN12 Yes No

Personal and Family History

Concept of Habit Index

The important aspects to be asked here are the habit patterns of the person, specially the abuse of tobacco, alcohol and any other drugs. It is important to note the frequency per day and length of the time that patient had the habit in years.

Habit Index

It is used in our department to quantity the effect of the habit.

For example if a person smokes 10 cigarettes for the last 15 years then the smoking index will be 10×15 = 150 (see Fig. 1.3).

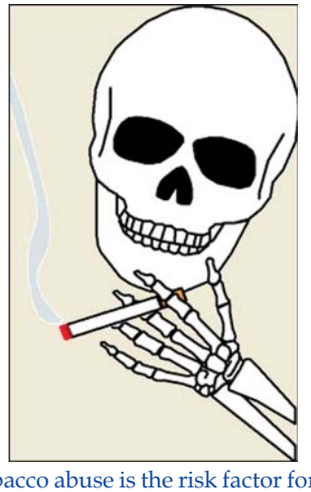


FIGURE 1.3: Tobacco abuse is the risk factor for many oral and systemic diseases. It needs to be recorded accurately (Bailoor DN, Keerthilatha Pai 2004)

Alcohol consumption usually is measured in peg per week × no of years, for example if a person consumes 2 pegs of whisky a day for ten years then his alcohol index will be calculated 14×10=140.

We divide the alcohol again into three categories.

- Risk one is Wine and Beer
- Risk two is Rum, Whisky, Gin etc.
- Risk three is Country alcohol, Arrack etc. (see Fig. 1.4).

The above example now becomes 140 risk two.

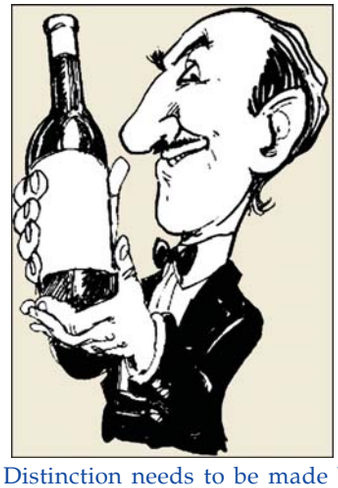


FIGURE 1.4: Distinction needs to be made between social drinking and alcohol abuse (Bailoor DN, Nagesh KS 2004)

Betel chewing, betel leaf chewing with slaked lime and catechu could also be quantified in similar fashion by a product of the frequency per day × no of years, at the frequency of 8 a day for twelve years of betel chew index would be =12 × 8=96

Record the frequency of tooth cleaning, method of tooth cleaning, whether indigenous or modern, uses of dental floss, mouthwash or any other modalities.

Details of the diet are asked specially if patient has any food fads, is a pure vegetarian, etc.

A family tree is drawn up, usually with father mother and diagram of siblings if any inherited disease is suspected, and the details of the members affected is duly recorded.

For example – Diabetes, hemophilia, hypertension, cleft lip, etc.

Fear of the dentist and his drill is almost proverbial. Dentistry today is painless and comforting. See that your patient feels comfortable and alleviate his fear to get good treatment compliance from him. All are afraid of dentists—remember that so your approach can be more sympathetic (see Fig. 1.5).

FIGURE 1.5: Fear of dentists or dental treatment is termed as odontophobia. Patients fear the dentist's injection and drill (Bailoor DN 2004)

Social and Occupational History

The fact that psychosocial factors affect the general health of the patient and his oral health is well established. So recording whether the patient stays alone (Loneliness) or in joint family (Intra-family tensions) becomes important. A woman may have mother in-law problem in her MPDS diagnosis!

Occupational stress can play a major role in lifestyle diseases of today characterized by Worry, Curry and Hurry

FIGURE 1.7: Stress is a major cause in grinding of teeth (bruxism), TM joint problems, Ulcers in the mouth and many other diseases (Bailoor DN, Nagesh KS 2004)

FIGURE 1.6: Showing mechanical abrasion on the crown of central incisors due to hold of bolts and nuts by car mechanic who reported with severe pain in the upper anterior region (Ajay Nayak, Prasanna Kumar, Bailoor DN 2004, Yenepoya Dental College and Hospital, Mangalore)

(see Figs 1.6 and 1.7). Cardiovascular diseases, headaches, hypertension, ulcers in the mouth and stomach, Sleeplessness and fatigue can all be a serious risk factor for the dental patient.

Bailoor DN and Nagesh KS 2004 have suggested a more holistic model for disease which takes into account the biological, psychological, spiritual and sociological factors. This model may be termed as the Bio-psycho-socio-spiritual model of illness. The findings to support this model were presented at the XIV national conference of the IAOMR at Hyderabad in December 2003 (Fig. 1.8).

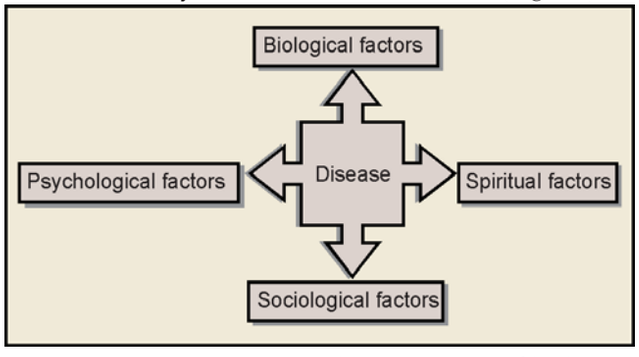


FIGURE 1.8: Diseases are caused by interaction of biological, psychological, social and spiritual factors. Holistic model of illness (Bailoor DN, Nagesh KS 2004)

Where the patient works, and what are his work tensions, affect of the important facets on his health. Now there are newer specialization like sports medicine and occupational medicine, which gives us good insight into this aspect of diagnosis.

Liss GM et al[8] (1997) have clearly indicated the importance of the occupational history in looking at newer diseases emerging in the clinics today. They also mentioned that hospital records that are properly codified and indexed are a good source of occupational risk information.

Jackson JL et al[9] (1998) have found four clinical clues that predicted patients likely to have depressive and anxiety disorders. They were Stress (recent); Somatic Symptoms; Status of health (generally poor or perceived by patient as poor); Symptom severity. They term it the 4-S way of testing.

The health psychology and its study today indicate that all the diseases today including oral diseases have what is termed as the bio-psycho-social etiological frames of reference. Lennart L[13] (1997) has clearly supported the biopsychosocial approach to etiology and pathogenesis when he indicates that emotions, behavior, stress, coping and social and family support play a great role in prognosis of a disease.

It is important to record the finding in a card or file and at the end of his statement, take his signature in presence of a witness. This helps us.

1. To enter changes that the patient may tell at a later date.
2. To protect ourselves in event of a medico-legal problem

EXAMINATION OF THE PATIENTS

Now we start examining the patient in this order, the general examination, the extraoral examination and the intraoral examination.

General Examination

Here the build, nourishment, consciousness and the cooperativeness of the patient are noted.

- Build—Well-built, moderately built or poorly built indicates the bone structure of the patient.
- Nourishment—Well, moderate and poor indicates the soft tissue profile of the patient.
- Conscious or unconscious—In dental OPD most of the patients will come conscious. Only in trauma or emergency care center will the patients be brought in stretcher.
- Note whether the patient is cooperative or not.

The weight, height, temperature, respiratory rate and gait of the person are recorded.

FIGURE 1.9: Using the BP Instrument is a must in any dental clinic. All obese patients and all patients above 40 years of age must be examined using a sphygmomanometer (Kiran K, Beena K, Bailoor DN 2004, Yenepoya Dental College and Hospital Mangalore)

Weight of the patient is recorded in Kg. Height is recorded in Meters and BMI is recorded by the formula,

$$BMI = \frac{\text{Weight in Kg}}{\text{Height square in meter}}$$

BMI is a clinically usable nutritional parameter by dentists. Either a manual or electronic sphygmomanometer records blood pressure, routinely, pulse and temperature is noted. Cyanosis, clubbing, pallor, any apparent lesions on the skin of the forearm, legs, etc. should be observed. A general dental practitioner need not routinely do cranial nerve examination but if he suspects any neurological deficit he must be able to express suspicion as to which cranial nerve is involved. Reference to a neurologist is usually a good idea in such cases (Fig. 1.9).

Extraoral Examination

- Eye—Spectacles, contacts, change in vision, inflammation lacrimation, color (Pallor, Jaundice, etc)
- Otolaryngological points—Pain in the ears, hearing changes, tinitus, sinus disease, mucous discharge, blood discharge, nasal obstruction, voice changes, sore throat and tonsillitis. The symmetry of the face. Overlying skin, bruising, itching and rashes. Observe for tremors, convulsions, anesthesia, paresthesia and paralysis (Figs 1.11 and 1.12).

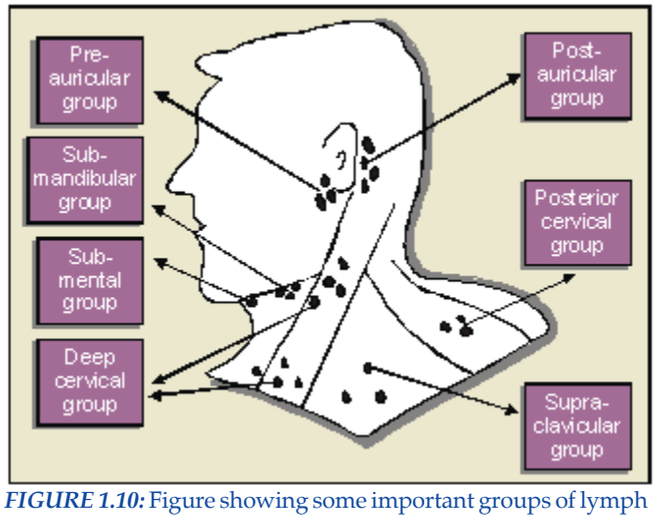


FIGURE 1.10: Figure showing some important groups of lymph nodes that a dentist must routinely palpate and then write a report on the number, consistency, tenderness, etc. of the lymph nodes (Prasanna Kumar, Bailoor DN, YDC Mangalore 2004)

Inspection

Of the face involves the observation of the symmetry of the face, swelling, how patients opens and closes, and if he is suffering from any tics, facial weakness, birth mark, etc.

FIGURE 1.11: Showing the deviation of the TMJ due to Fibrous ankylosis on the right side. The right TMJ will be affected in this case. (Prasanna K, Beena K, Bailoor DN 2004, Yenepoya Dental College and Hospital, Mangalore)

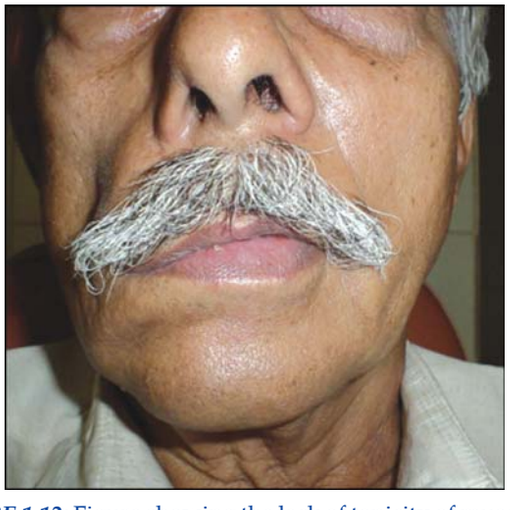
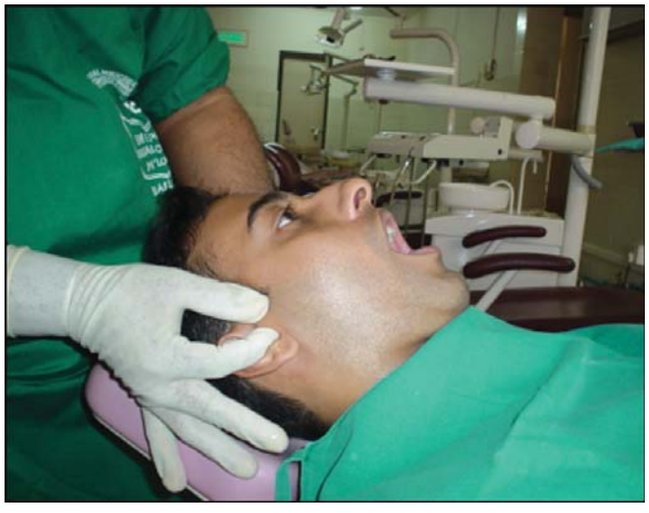


FIGURE 1.12: Figure showing the lack of tonicity of muscles in the right side of the face with drooping of the angle of the mouth on smiling in patient of Bell's Palsy. (Prasanna K, Beena K, Bailoor DN 2004, Yenepoya Dental College and Hospital, Mangalore)

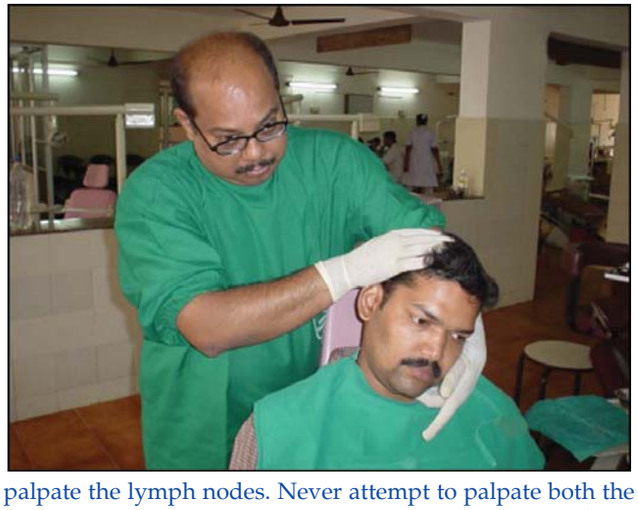


FIGURES 1.13A and B: Figure on the left showing the TMJ being palpated by the two finger method—doctor positioned posteriorly . On the right it shows the doctor positioning from the anterior aspect. Here the clinician can observe even slight deviation (Nayak A, Bailoor DN 2003. Yenepoya Dental College and Hospital, Mangalore)

Palpation

A regular palpation of TMJ and the lymph nodes in the cervical and the peri-oral regions is mandatory. If any swelling, asymmetry or obvious deformity is evident, clinically palpating and recording the size, shape, consistency, fixity to underlying regions, and other properties must be recorded (Fig. 1.10).

The temporomandibular joint (TMJ) is palpated using either the one-finger method or the two-finger method. The dentist may position himself either in front of the patient or behind the patient. We advocate the TMJ palpation with a two-finger method in our department. The forefinger is inserted in the external auditory meatus gently and ball of the thumb is placed on the preauricular region to feel for the clicks, popping, crepitus, and tenderness. The patient is asked to open and close the jaw gently; the degree of opening and deviation if any is noted. The patient is 2approached from the front with his chair position being



FIGURES 1.14A and B: (**A**) Wrong way to palpate the lymph nodes. Never attempt to palpate both the sides at the same time. The patients neck is stretched and this will preclude the early detection of any changes in the consistency of the lymph nodes. (**B**) Right way to palpate the left submandibular lymph nodes by tilting the patients head on the same side (Prasanna K, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

upright. A stethoscope could be used to amplify the TMJ sounds if there are any positive findings (Fig.1.13).

Lymph nodes of the submental, submandibular, pre-auricular, post-auricular, superficial and deep cervical group are palpated and recorded as palpable/non-palpable, tender/non-tender and the quality like matted, hard rock like, rubbery, etc. are recorded so that their clinical significance could be integrated with the final diagnosis (Fig. 1.14).

Bi-digital palpation of the floor of the mouth region is an important component of any lesion in this area and for early detection of sialoliths in submandibular gland ducts.

Facial skin and facial symmetry should be noted for any abnormality, angle of the lips for any lesions like angular cheilitis, ulcers like primary herpetic stomatitis.

Nasolabial fold should be consciously observed and its obliteration may be seen in Bell's palsy, or sometimes swelling in the canine region of the face.

- Intraoral examination again is divided into soft tissue analysis and hard tissue analysis.
- Soft tissue should be examined thoroughly especially, at the ventral portion of the tongue, the floor of the mouth, the tonsillar fauces examined in addition to the buccal mucosa, plate, labial mucosa, etc.
- The lesions like white lesion, vesiculo-bullous lesion, pigmentations, ulcerative lesion, etc. should be noted.
- Hard tissue analysis—Usually a notation of decayed; missing and filled teeth is made on each tooth examined. The caries is further classified as occlusal, proximal, or smooth surface and root according to location. It is important to note whether the caries is primary, secondary, or rampant according to distribution. The qualifying words are used wherever relevant. The regress ional changes such as attrition, abrasion and erosion also are duly recorded.

We use a visually appealing dental record for initial noting of the conditions as shown in Figure 1.15.

KEY

Decayed	D	Missing	M
Attrition	AT	Filled	F
Abrasion	AB	Root stumps	RS
Erosion	ER	Crown	C
Mobility	MO	Bridge	B
Furcation involvement	FI	RPD	RPD
Fracture	#	Pulp Exposure	PE
Discoloration	DI	Pain on Percussion	POP+/-

Tentative: Diagnosis is now recorded by describing the positive finding in the above examination. It states the sex, medical status, soft tissue diagnosis and hard tissue diagnosis. For example a typical tentative diagnosis would read; A 45 -year-old male diabetic (6 years) on treatment, with generalized suppurative periodontitis and caries in relation to 36 and 46.

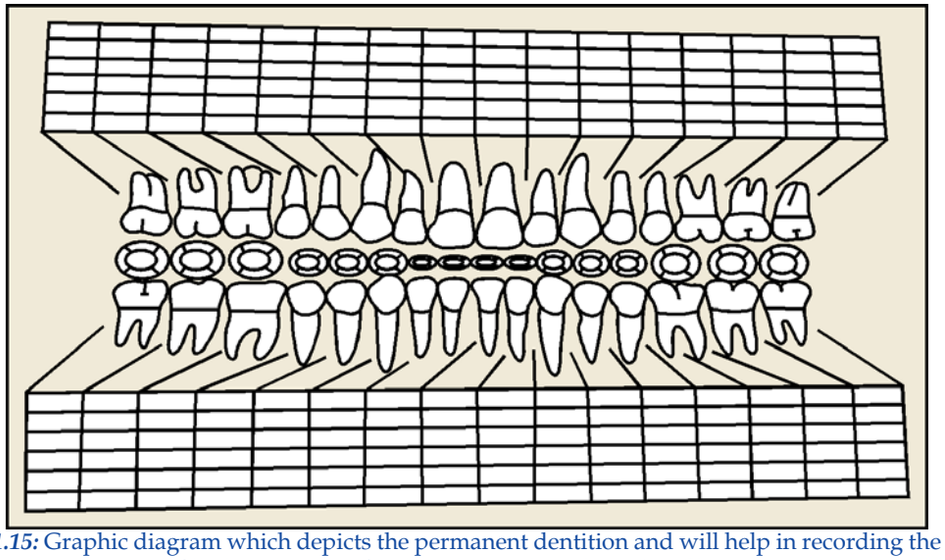


FIGURE 1.15: Graphic diagram which depicts the permanent dentition and will help in recording the decayed, missing, filled teeth status for dental record (Bailoor DN, Chatra LK 2004)

- After tentative diagnosis usually the dental surgeon asks for some tests to be done. If the patient has some metabolic or systemic problem then routine hematology and urine testing usually gives an important clue to follow up. If some soft tissue lesion is there, then usually a biopsy, of incision type is performed, the bit of tissue fixed in 10% formalin, and then a histopathological testing is done. In case there is gross carious destruction or advanced periodontal disease or any other hard tissue involvement then the best test to be performed is the Intra oral peri-apical radiography or the IOPA.. (See the diagnostic sequence chart) or screening-radiograph usually preferred is the Orthopantomograph (OPG) See chapter no 29. As the results from these tests come through a positive confirmation name of the Lesion emerges. This then would be termed as the final diagnosis.
- Normally treatment plans are based on the final diagnosis. Treatment plans are charted in many ways, in our department we use the following chart. This chart is divided into dept. Sections merely to facilitate the divisions of work and to do time management. It also helps us to prioritize the treatment keeping in mind the chief complaint of the patient.

Medical Alert:			*Allergy Profile*	
Appointment	Physicians Referral	Oral Medicine	Radiology	Periodontia
Appointment	Restorative	Oral Maxfac Surgery	Prosthodontics	Orthodontics

Two other columns could be added to this for noting time and date of the appointment given and also the charges that are charged on that particular day, this can be again cross-referenced with the financial accounting done at the clinic.

Kay and Nuttal[12] (1995) make an important point about assessing the risks involved in all the treatment plans and determining the probabilities of success in various treatment options. Using the concept of Evidence Based Dentistry (EBD) clinician assesses the risks. The clinician does a thorough examination of peer reviewed literature. He then communicates clearly to the patient the risks and benefits of the procedure in order to involve him in the decision-making process of the treatment planning.

Today it is recommended that the entire record keeping should be done on microcomputer system together with a good quality printer, this will make the dental surgeons job much easier and more accurate.

Sicotte C et al[11] (1998) state that reengineering of the workplace through Information Technology is an important strategic issue for today's hospitals. The Computer-based patient record (CPR) is one technology that has the potential to profoundly modify the work routines of the care unit. It also raises ethical and confidentiality related problems. Szekely DG et al[4] (1996) have highlighted how human errors as well as software design errors can impinge on clinical data security.

Warren JR et al[10] (1998) mention about the Patients Interview Support Application (PISA) which is a program intended for operation by a non-expert clerk to interview an ambulatory primary care patient. This program was downloaded on to the web. The resultant Web environment attracted thought-provoking and detailed feedback from users, indicating that significant attention can be obtained from the global community by mounting an interactive system on the Web. Specific enhancements to the PISA's artificial intelligence are suggested by user reaction. These authors envision a future global health informatics 'marketplace' with a multitude of Web-based system components available for composition of health information systems.

- See the module on Computers in Dentistry chapter no 30 for further details.
- Problem Oriented Recorded (POR) keeping have also become popular in some specialties where each problem of the patient is recorded and its detailed resolution planned therapeutically before going to the next.

SUMMARY

History taking, clinical examination and the investigative tests make a good diagnostic sequence.

Correct selection of tests is important for proper treatment planning. Treatment planning takes into account the principal reason that the patient came to you, his attitude, his medical status and finally his financial status.

Indian income tax Act also mandates that a form 3C be maintained by all dental surgeons in which the patient's name, treatment rendered and fees charged be recorded on a daily basis.

Take help of a professional chartered accountant to help you in maintaining and filing the tax returns every year.

Accurate recording system helps to do good treatment, remember financial details and protects you from any consumer or legal action, which may arise due to some misunderstanding by patient of your treatment decisions.

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2 Systemically Compromised Dental Patients

INTRODUCTION

With the advent of miracles of modern medicine, the strong pharmacological agents, the pacemakers, the dialysis units and the digital imaging, more and more dental patients with serious medical compromise are likely to walk into dental clinic. It is a practicing dentist's duty to recognize such medical deviations and then take treatment decisions.

In a Dental Hospital in Mangalore, the data on 11784 patients were analyzed by Bailoor DN, Gopakumar et al (1991).[5] They concluded that 7.7% of the patients had medical compromise. Each Medical problem was codified during the initial patient data entry itself or after receipt of relevant laboratory inputs.

The commonest systemic disorders affecting the dental patients in South India were determined to be

1. Atopic conditions 39%
2. CVS 24%
3. Diabetes 11.07%
4. Respiratory 10.09%
5. Neurological/Psychiatric 3.36%
6. Oral cancer 1.9%
7. Pregnancy < 1%
8. Miscellaneous group orthopedic, hepatic and undefined medical compromises constituted around 9% of the disorders.

ATOPY IN DENTAL OFFICE

The following were allergy related conditions seen

1. Stomatitis medicamentosa (angioedema)
2. Stomatitis venenata
3. Serum sickness
4. Anaphylaxis

Atopic disease is a name given to group of allergic conditions. This disease is mediated by specific IgE antibody, which binds to the mast cells. Further exposure to an allergen results in degranulation of the mast cells with release of mediators of allergy such as histamines.

Tests

Some of the tests done in Atopy are as follows:

Skin Test

a. Pricking the allergen into the skin and waiting for the wheal to appear.
b. Applying allergen into the skin by an absorbent dressing material – termed as patch testing.

Laboratory Tests

a. Serum IgE levels determination by PRIST (paper radio-immunosorbent test)
b. RAST—Radioallergosorbent test for IgE antibodies to specific antigens. Stomatitis medicamentosa is an old

term, used by some senior professionals. It was used to denote systemically mediated atopic reactions to some dental products or drugs. Today most oral medicine specialists prefer to use atopic reaction or angio-edema in such cases. The most common manifestation is a swelling of one or both lips acutely, facial and neck swelling, occasionally serious enough to cause respiratory embarrassment.

Stomatitis venenata or contact allergic reaction is commonly seen to silver amalgam, methylmethacrylate denture base, eugenol, toothpastes, and mouthwashes. Good history and alert clinician will be able to diagnose such allergies and treat them.

Rare condition termed as hereditary angioedema has been observed in some dental clinics. Hereditary angioedema is an autosomal dominant disorder resulting from the deficiency of the C1 esterase inhibitor. Generalized facial edema , edema of extremities , abdominal pain and vomiting are characteristic of this condition. This may be precipitated by blunt injury , dental treatment or stress. The treatment for this condition is done using tranexamic acid and drugs like stanazolol (2.5 to 8 mg daily). Farkas et al (1999)[15] evaluated the efficacy of danazol(600 mg/d) treatment on 12 dental surgery patients. He has established that this drug prevented all the patients from showing any complications of the hereditary angioedema. In all the patients the serum levels of the complement components were decreased immediately after surgery and returned to normal within 24 hours.

Atopic reaction was seen to:

1. Penicillin
2. Sulfonamides
3. NSAIDs (Ibuprofen and Flubiprofen) as three main groups of medications involved.

Those with low allergic potential are:

1. Erythromycin
2. Tetracyclines
3. Lidocaine
4. Digitalis
5. Acetaminophen.

Stomatitis venenata was observed to impression materials, denture base, and mercury from amalgam filling.

Management

It includes identifying and discontinuing the causative agent. Following antihistaminics were used with very good result in such conditions.

- Astelong® 10 mg (Astemizole) 'Torrent' one tablet once daily increasing upto three tablet a day.
- Avil® tab 25.50 mg Syrup (Pheniramine maleate 22.5 mg and 45 mg ; 'Hoechst'® 25 mg tds or 50 mg bd).
- Polaramine® tab, syrup 2 mg (dexchlorpheniramine maleate) 'Fulfor' one tab adult tds child + tab tds
- Phenergan® 10 mg day.
- Foristal® 1 mg (dimethindene maleate) 'Hindustan Ciba Giegy' one tab tds for a week at least.

Severe allergic reactions could be treated with 60 mg prednisolone daily in divided doses to be tapered down to 5 mg. In a period of two weeks, Aminophylline is often used in the beginning stages to relieve Bronchospasm together with intermittent use of oxygen mask. Life-threatening allergic reaction is best treated with 0.5 to 1 ml of 1:1000 aqueous adrenaline subcutaneous. Here oxygen intubation is imperative.

The Serum sickness and Anaphylaxis are dealt in the chapter no. 6 Medical Emergencies.

CARDIOVASCULAR SYSTEM AND THE DENTAL PATIENT

In our series 23.8% of the patients with medical risk had this problem.

We divide the CVS problems into two main groups**.**

In the dental clinic:

1. Those disorders which require antibiotic prophylaxis
2. Those that do not require prophylaxis.

*Congenital heart diseases***:** Rheumatic carditis, Valvular heart diseases, etc. require prophylaxis.

Congenital heart disease occurs in 0.5% of all live birth (Rose and Kaye)[1] common examples being ASD, VSD, pulmonary stenosis, over-riding aorta, etc. It is important for the dental surgeon to have a written prior permission from the cardiologist before instituting any dental treatment. In patients, known to have this problem, regimen A of the American Heart Association is recommended (See Table 2.1).

Aquired heart disease/coronary heart disease: No antibiotic coverage is required unless the local infection warrants its use. Patients should be advised to bring tablets like Sorbitrate 10 mg (Isosorbide dinitrate) with them so that in the event of pain in the dental chair, the tablet could be at once, administered. Dental surgeon could keep amyl-nitrate, which can be crushed and inhaled, in dire need. Nitroglycerine is now available in a gel like matrix attached to an adhesive bandage that delivers the drug intra-dermally, the bandage is effective for 24 hours. For long-term therapy of Angina calcium slow channel blockers like Nifelat 5 mg, 10 mg capsules (Nifedipine) 5 mg tabs are recommended. In exceptionally apprehensive patients, 2 mg diazepam for emaciated patients below 50 kgs and 5 mg diazepam for those above 50 kg is recommended this may obviate the use of antiangina medication. Patients of MI are usually on anticoagulant therapy. Dental surgeon should not make any attempt to reduce or alter the regimen.

Normally if the patient's prothrombin time and partial thromboplastin time are within therapeutic range it should be possible to carry out most of the procedures without altering the patient's usual dose. If the dose has to be reduced then patient's physician should be directly involved and procedures are done in hospital setting where adequate postoperative nursing is available.

Hypertension2,3: Successful management of hypertensive patients depends on early recognition of first time cases, on good pain control, and prevention of postoperative hemorrhages. Dental surgeon must routinely record blood pressure of all dental patients and specially keeping in mind the high risk group. This includes the patients who are:

1. Obese
2. Pregnant
3. Tense and anxious
4. Diabetic
5. Any one with throbbing pain and headache
6. Age above 45 years.

A single raised value does not indicate hypertension but three consequent values taken more than a week apart should make the clinician suspicious. The following guidelines for mild, moderate and severe may be followed.

Muzyka BC et al [6] mention that dentists must be able to recognize risk factors associated with hypertension and counsel patients in addition to taking care to see that none of the complications rear their ugly head in the clinic.

Diastolic 90-104 (Mild)
105-114 (Moderate)
115 and above (severe)

Systolic 140/159 (Moderate)
Above 160 (severe)

Jastak et al[3] clearly mentions that it is acceptable to use vasoconstrictors in patients with mild to moderate cardiovascular disease, however in severe cases which are hospitalized LA free from epinephrine was suggested for example in poorly responding coronary heart disease, life-threatening arrhythmias etc.

Lynch MA[4] says that his experience and observation is that epinephrine in the LA contributes to good local hemorrhage control and does not significantly alter the BP. There is no sufficient reasons for a private practitioner to use adrenaline free LA.

Rheumatic heart disease and bacterial endocarditis: In these conditions clear-cut antibiotic protection is suggested and Regimen A is recommended (See Table 2.1).

Latest recommendation for antibiotic prophylaxis: Langlais RP and Miller CS (1998)[23] for the dental patients undergoing invasive dental treatments.

DIABETES MELLITUS (DM)

Diabetes mellitus (DM) is caused due to absolute or relative deficiency of insulin. Two main types—the juvenile onset and the maturity onset—type of DM should be kept in mind by the practicing dentist. The juvenile DM dental patient would typically be having family history and be within 25 years of age. Recent loss of significant amount of body weight should alert the dentist. Weakness and fainting spells in high school and college are frequently mentioned in the history.

Maturity onset DM patient is typically in his mid-forties, family history positive, sedentary mode of occupation and slightly or really obese. Two kinds of patients would be seen in the dental clinic

a. Not a known diabetic but dentist suspects due to history and clinical examination.
b. Established diabetic under treatment of the physician.

Table 2.1

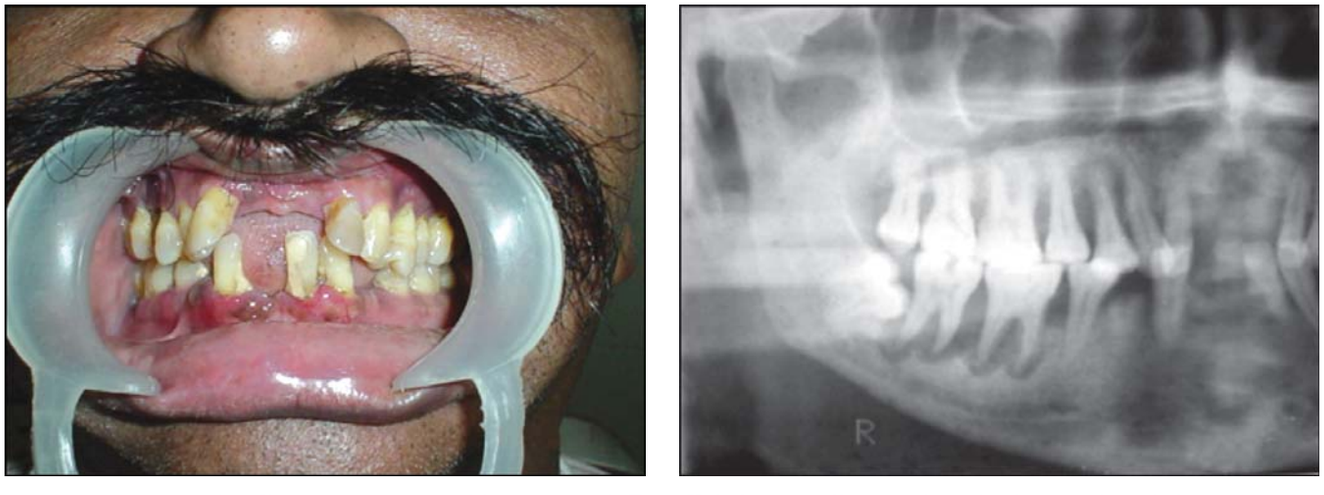
ADULT DOSES		
2 gm oral amoxicillin Allergic to penicillin	1 hr before dental procedure	1 gm 6 hr after the treatment
Oral clindamycin 600 mg	1 hr before dental procedure	300 mg 6 hr after the treatment
Oral Azithromycin 500 mg	1 hr before dental procedure	No repeat dose needed unless specified by phycisian
CHILDREN DOSES		
Amoxicillin Elixir 250 mg/5ml	Less than 15 kg – 750 mg 15 - 30 kg – 1000 mg above 30 kg – 1500 mg	50 mg/kg body wt 1 hr before and 25 mg/kg body wt 6 hr after the procedure
Clindamycin	20 mg/kg body wt 1 hr before	10 mg/kg body wt 5 hr postopertive
Clarithromycin	15 mg/kg body wt 1 hr before	Same dose 6 hr postoperative
WHEN IN DOUBT CONSULT AND GET WRITTEN CONSENT FROM PHYSICIAN		

Suspected diabetic: If a dentist looks at severe periodontitis (disproportionate to the local factors), partial dryness of mouth, burning tongue or persistent periodontal abscesses, he must send such a patient for Glucose Tolerance Test (GTT) to a nearby lab. If patient shows positive on the GTT then immediate physician referral is indicated. No dental treatment is indicated in patients with uncontrolled DM status. After a written physician's consent is obtained only then should any kind of the treatment be initiated.

Known diabetic under medication or treatment: Request for a random serum glucose or accept a report which is within last 48 hours. Record the physician's name and telephone no. and call him prior to initiating any major dental surgery. DM patients have increased propensity to post-operative infection so bactericidal antibiotic therapy is indicated at least for a period of five full days after extraction or any other surgery. When in doubt get written consent from physician and keep him informed.

Oral manifestations of DM have been reported as:

1. Severe periodontitis disproportionate to the local factors
2. Persistent suppuration in various parts of perio-dontium
3. Oral candidiasis
4. Partial Xerostomia
5. Burning tongue
6. Sialadenosis
7. Lichenoid reactions secondary to oral hypoglycemic drugs (see Fig. 2.1).



FIGURES 2.1A and B: Showing a 46-year-old patient with Type II diabetes mellitus with multiple periodontal abcesses and horizontal bone loss generalized (Prasanna K, Bailoor DN 2003 Yenepoya Dental College and Hospital, Mangalore)

In their study of 414 insulin-treated diabetic patients, Willis AM et al (1999)[10] have categorically stated that 77% of the patients had Candida organisms which could be isolated from their oral cavity, the commonest clinical finding was that of erythematous candidiasis. The incidence of this kind of candidiasis was significantly associated with smokers and those who wore dentures.

A triad of findings of DM, oral lichen planus, and hypertension has been termed as Grinspan syndrome but many researchers around the world today believe it to be coincidental.

As a general rule in brittle or uncontrolled DM cases the dental treatment should not be done in the dental clinic and are better treated in the wards of dental teaching hospital or a general hospital with consulting oral medicine and oral surgical specialists.

A diabetic patient who is well controlled can receive regular dental treatment. In brittle cases it is best to schedule appointments in mid-morning following the patients breakfast and normal calorie intake through soft food and liquid diet otherwise hypoglycemic shock would result. Infection being a routine complication antibiotic cover with erythromycin is ideal at least five days after the operative procedure. In the event of patient undergoing shock in the dental chair 2% glucose IV is advised till the physician comes. If the vein is difficult to find, 1 mg of glucagon IM can be given.

RESPIRATORY SYSTEM DISEASES

In this group of diseases the upper respiratory complaints like pharyngitis, tonsillitis and laryngitis are easily treated and usually do not complicate the dental treatment. Chronic sinusitis, however, often results in dull and ill-defined pain in the maxillary posterior segment and the absence of local pathology confound the dental surgeon about the diagnosis. In some patients the primary complaint of halitosis is easy diagnose and treat. See chapter on Maxillary Sinus Pathology for detailed discussion.

In the lower respiratory group asthma and tuberculosis are of importance to the practitioner. In asthma the local treatment of bronchospasm is given in Figure 2.2.

In the dental clinic an inhaler like Bakelite® inhaler (Cipher) containing Beclomethasone Dipropionate 50 microgms/inhalation can be kept handy and is of life saving importance in any aggravation.

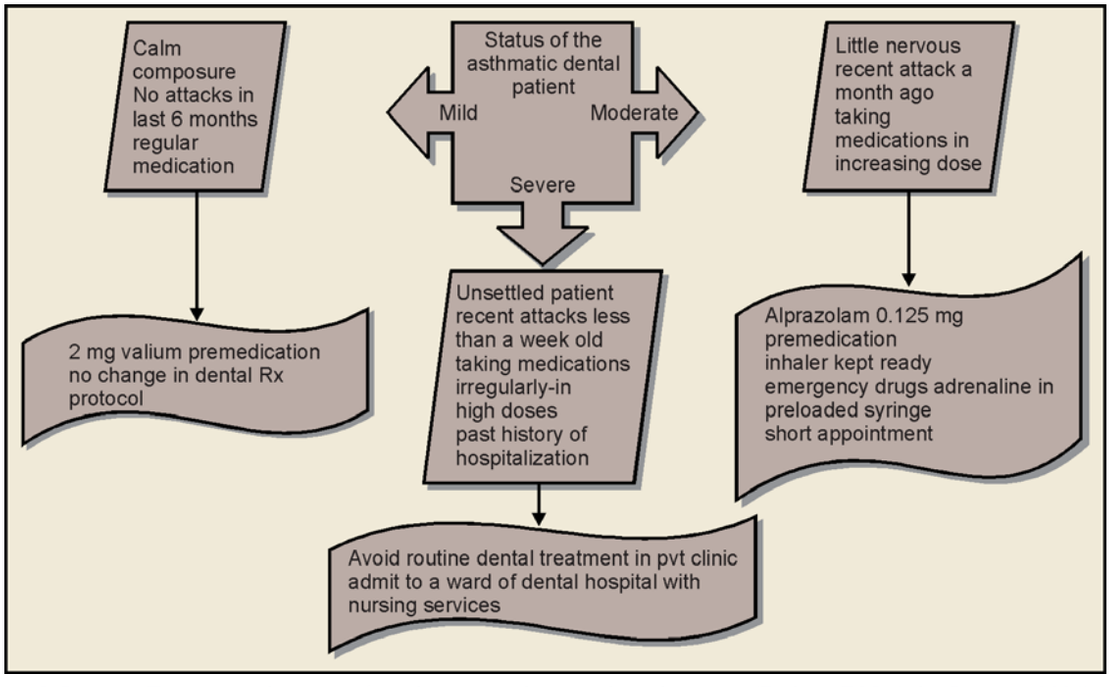


FIGURE 2.2: Treatment decisions for a known asthmatic dental patient (Bailoor DN, Asha Iyengar 2004)

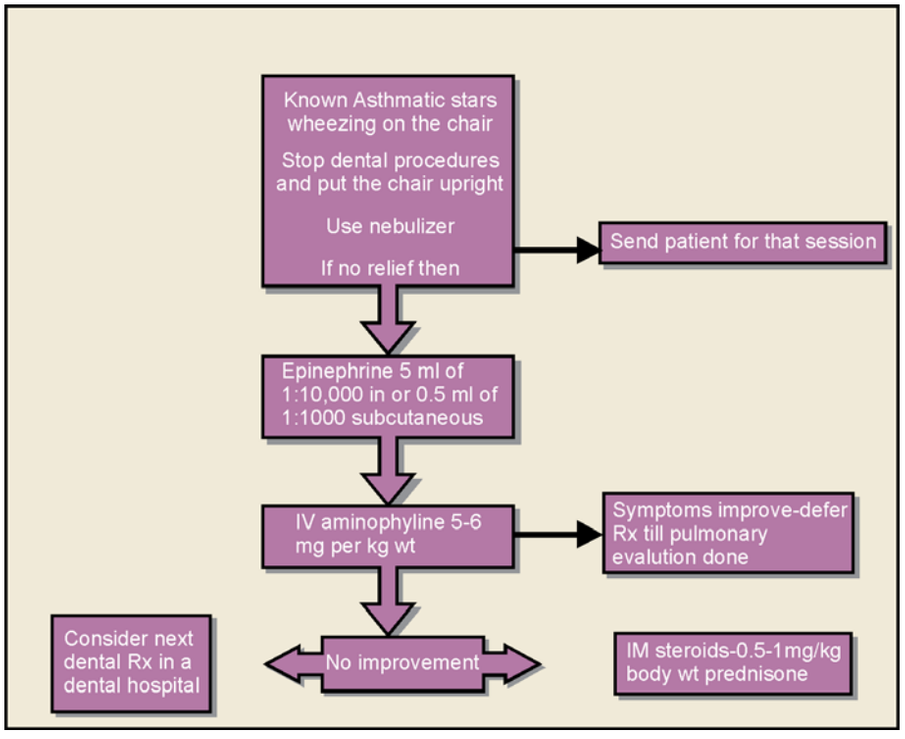


FIGURE 2.3: Treatment of asthma on dental chair (Bailoor DN, Nagesh KS, Asha Iyengar 2004)

The asthmatic patients who attend dental clinic need to be classified as Mild, Moderate and Severe risk.

Mild— Those who have history of asthmatic attacks in the past , no recent attack in last six months, stabilized on medication. Regular dental protocol, with 2mg Valium half an hour prior to the dental treatment to relax the patient.

Moderate—Recent asthmatic attack less than four weeks old, and patient appears nervous and uncomfortable, keep inhaler ready by the side, oxygen mask and nurse aid to be alert or if not available regularly to be called in, pre-medication with 2 mg Valium is a good idea.

Severe—Asthmatic attack as recent as a week old, high levels of medication, past history of hospitalization due to asthma. Do not treat in dental clinic, but post this patient in the wards of dental hospital with round the clock nursing available and all the emergency drugs available at hand (see Fig. 2.3).

Bang LM and Plosker GL [25] have outlined treatment with Omalizumab® (Xolair) is a humanized monoclonal antibody used in the treatment of adolescent and adult patients with moderate to severe allergic asthma inadequately controlled with inhaled corticosteroids (ICS). It selectively binds to circulating immunoglobulin E (IgE) and, thereby, prevents binding of IgE to mast cells and other effector cells.

Tuberculosis dental considerations: Any dental patient who is detected with classical signs of pulmonary tuberculosis in India today should be immediately sent for ELISA test for HIV to the nearest center in addition to the control of lung infection. Extra precautions regarding cross infection are a must and the oral manifestations include chronic ulcers on tongue, granulomas and cervical lymphadenopathy (cold abscess)

Junquera GLM et al (1996)[9] have reported a case of primary tuberculosis in the oral cavity the ulcerative lesion of which developed in a recently extracted tooth socket. The bacteria M tuberculosis hominis was identified microbiologically. They acknowledge in this report that the primary TB in oral cavity is rare.

Stelianides S et al (1997)[7] found that immunodepressed patients notably those infected with HIV are particularly prone to a polyvisceral tuberculous infection. The most frequent localization are the lymph nodes. Confirmed diagnosis always rests on histological and/or microbiological evidence

De Aguiar MC et al (1997)[8] reported Pulmonary TB patient who presented with multiple oral ulcerations with an irregular periphery and a granular vegetative fundus.

Patients of (COPD) chronic obstructive pulmonary disease are usually contraindicated for the General anesthesia and most treatments should be planned for in local anesthesia keeping oxygen mask ready is a good idea in case of distressed breathing attack. Prior physician's fitness should be asked for and kept on file.

The upper respiratory system diseases may present as halitosis and dysphagia as presenting symptoms and the serious lower respiratory systems are recognized by their specific signs and symptoms and most of the dental management may be attempted in the hospital set up.

THYROID DISORDERS AND DENTAL IMPLICATIONS

Dental surgeon may routinely find nodules in the thyroid while doing extraoral examination. What he needs to determine is whether the patient has euthyroid or toxic thyroid. The toxic thyroid usually results in hypertension, increased body temperature and high pulse rate. The enlargements of thyroid are referred to as goiter and may be nodular or diffuse. If patient is taking any regular medication or seeing an physician then his written consent is mandatory prior to doing any radical dental treatment.

Endemic goiters are present in Himalayan and sub-Himalayan regions. Iodination of the salt has resulted in a significant reduction in this type of the goiter.

Dental considerations are that hypothyroidism may lead to large tongue (macroglossia) , delayed eruption of the mixed dentition , cold clammy skin and facial myxedema is seen. Small vessel bleeding is aggravated due deposition of subcutaneous mucopolysaccharides. Delayed wound healing is observed.

Hyperthyroidism patients show exophthalmus (protruding eyeballs) , early eruption of teeth in mixed dentition, increase bleeding due to hypertension and elevated heart rate (see Fig. 2.4).

GASTROINTESTINAL SYSTEM

The disorders affecting the gastrointestinal tract are multifarious and only some of the important and

FIGURES 2.4A to C: 60 years old female patient with multi-nodular non-toxic goiter. There is no absolute contraindication for total dental extraction in such cases. Patient was advised for surgical removal of thyroid for esthetic reasons (Prasanna Kumar, Nillofer Shabnam, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

commonly interacting ones are ; halitosis, xerostomia, ptyalism, dysphagia, gastritis, peptic ulcer, duodenal ulcer, inflammatory bowel disease, hepatitis, cirrhosis, End-stage liver disease, irritable bowel syndrome, diseases of the anus and anorexia nervosa.

Halitosis should be diagnosed into three varieties; local factors, systemic factors and psychological factors. The detailed assessment of halitosis is given in Chapter no 21.

Xerostomia (Dry mouth)

Xerostomia has many causes. Some of them may be classified as below (Fig. 2.5):

The protective effect of saliva in terms of washing off the food debris, and bacterial toxins plus the immunology aspect of protective lysoenzymes and IgA protection that it offers against the periodontal disease and the caries is well understood. Only when xerostomia sets in that the clinician appreciates the havoc it causes in the oral milieu and he must use compensatory mechanisms like use of artificial saliva, local application of topical fluoride solutions or gel on teeth and use of local antimicrobials like chlorohexidine mouthwashes to regulate the oral infections.

Dysphagia

This term literally means disturbed swallow. It is usually either acute or chronic. This problem when mentioned to the dentist must never be taken very lightly and must be investigated in very systematic manner. Detailed history, clinical examination all the lymph nodes, larynx and thyroid. Barium swallow, routine chest radiograph and fiber-optic esophagoscopy is to be routinely done. If any pathology is detected then the patient should immediately consult a gastro-enterologist. For detailed information about dysphagia, see Chapter 23.

Anorexia nervosa: It is a biopsychosocial disorder that commonly affects the teenage females who want to conform to some utopian ideal of thin body. The high pressure advertising, the urgency to look beautiful and the desperation for a positive body image all contribute to this disorder. It is characterized by the persistent vomiting and abhorrence for all kinds of food. This can lead to cervical erosions in most teeth and severe nutritional deficiencies. The dentist himself can do basic counseling but in most cases psychologist has to be involved in the treatment protocol.

Gastroesophageal reflux (GOR) , gastritis, peptic ulcer, carcinoma of stomach:

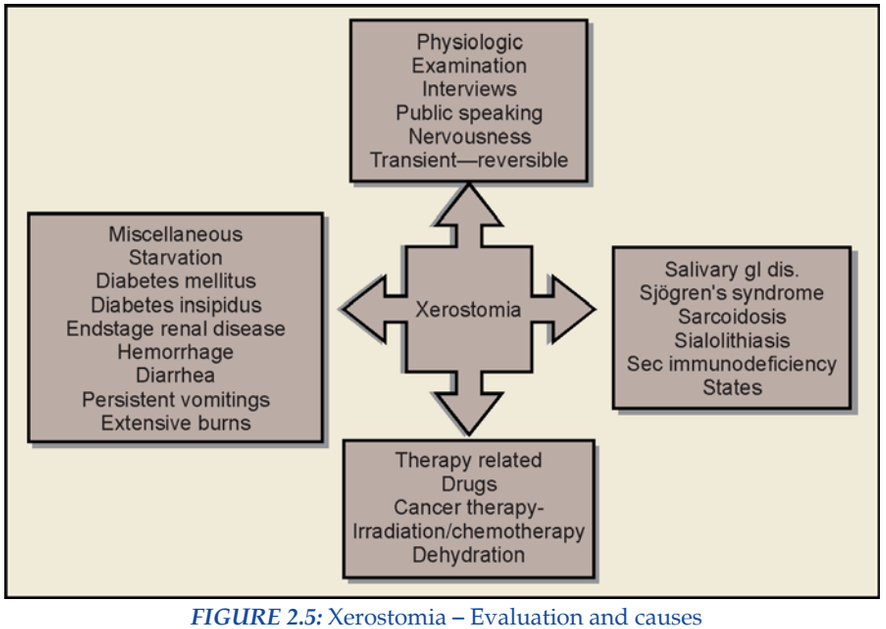


FIGURE 2.5: Xerostomia – Evaluation and causes
(Bailoor DN, Mukta Motwani, Mahima Patil 2004)

GOR normally occurs when the gastroesophageal sphincter becomes lax due to pathology or excessive insult by alcohol and spicy food. Persistent GOR can result in esophageal ulcers and transient dysphagia.

Bartlett DW et al (1997)[14] measured the oral pH using a radiotelemetry capsule incorporated in a palatal splint and found that there was a significant GOR – Gastro-esophageal reflux in patients who consumed alcohol and spicy food compared to persons consuming bland food without any accompanying drink. Thus, patients food consumption history together with occupational stress could hint at GOR and accompanying gastritis.

Epigastric pain when the hunger pangs hit, which are relieved by antacids or by food usually indicate different clinical possibility of either pre-ulcer gastritis state or frank ulceration. Physcians usually treat peptic ulcers by cemetidine, H_2 blockers, Bismuth chelates and by antibiotics to combat *Helicobacter pylori*. Dentist must not prescribe aspirin and other NSAIDs and corticosteroids.

Drugs like paracetomol and nimesulide have been considered safe for these patients. In fact a study by Gupta SK et al (1998)[24] indicates that nimesulide given by intramuscular route is potent anti-inflammatory and analgesic activity with no gastrointestinal side effects. They compared this with diclofenac injectibles and again found nimesulide favorable. Various studies have found nimesulide to be useful when administered orally, rectally or topically.

Diet counseling is mandatory by the dentist for such patients and decrease in alcohol consumption and spiciness of food should be considered primarily. Patients who may give history of vomiting blood , vague pains in epigastric region and feeling of lump in the stomach should undergo barium meal radiography and physicians examination to rule out carcinoma of the stomach.

Inflammatory Bowel Disease (Crohn's Disease and Ulcerative Colitis)

This is an inflammatory bowel disease of unknown origin. In some patients ulcerative colitis is reported as a side effect of some long-term antibiotics.

Rooney TP (1984)[11] reported that severity of caries incidence was higher in patients with Crohn's disease in his study of 21 patients. Halme L et al (1993)[12] again confirmed in their study of 53 patients that there were more infectious focii in teeth in an panoramic radiographic study. Nine of these patients with active disease also showed characteristic lesions of buccal mucosa on biopsy. Sundh B et al (1982)[13] mentions that strict oral hygiene and regular use of fluoride treatment is definitely justified in treatment of Crohn's disease patients.

The oral mucosal lesions associated include ulcers, labial swellings or cobblestone proliferation of the oral mucosa. In India tuberculosis and sarcoidosis should also be considered in the differential diagnosis. Dentist must keep in mind the fact that Crohn's disease patient would be suffering from malabsorption, corticosteroid treatment or immunosuppressive therapy.

Irritable Bowel Syndrome

This is a biopsychosocial disorder which is associated with a very anxious personality with a prevalence of upto 30% in most populations. The presenting symptom is severe abdominal pain, which is recurrent. This has been explained as being due to increased tone and activity of colon due to higher center overstimulation.

It is strongly associated with migraine, MPDS and other psychogenic disorders.

Minor tranquilizers (Valium, 5 mg) and high fiber diet should be the dentist advice to such patients before they seek specialist advice from clinical psychologists and gastroenterologist.

ANEMIA

Anemia is essentially reduction in oxygen carrying capacity of the blood caused by reduction in hemoglobin level below normal. Anemia is not a disease but one of the signs exhibited by lowered hemoglobin levels and the symptoms caused by it. Most common cause of anemia in India is iron deficiency, parasitic hookworms and malaria. Cultural factors in which women eat last and leftovers, together with regular menstrual blood loss and multiple pregnancies result in very high incidence of anemia in females.

Causes of Anemia

1. Increased demand for iron
2. Excessive loss of blood
3. Increased destruction of RBC (Red blood cells)
4. Bone marrow depression and decreased production of RBC
5. Decreased absorption of iron
6. Drugs and some medical compromises.

Clinical Features

In many patients no specific features are noted in mild anemia. As the level of Hb drops there is a general feeling of weakness, distressed breathing, angina, and murmurs. Nails show pallor, brittleness, and spoon-shaped abnormality (see Fig. 2.6).

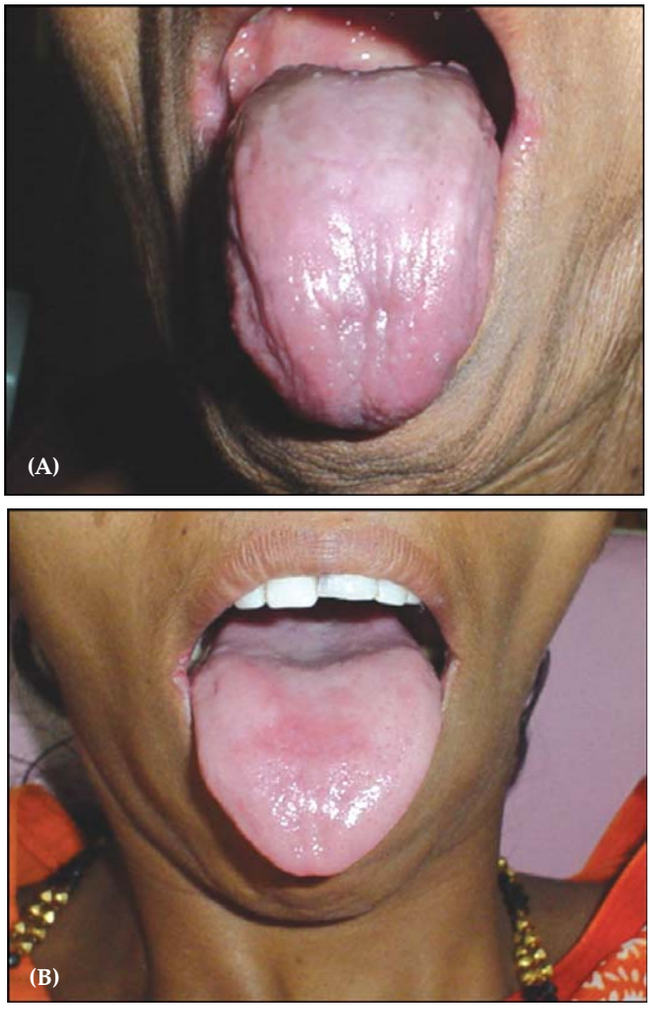
In the oral cavity mucosa appears pale, tongue appears bald and sometimes red and inflamed. Oral ulceration, angular cheilitis and burning sensation are seen in most patients. Iron, folic acid and vitamin B_{12} are usually given together for management of nutritional anemia.

In India, parasitic infection by hookworms and malaria are the most common cause of anemia. The first is linked to unhygienic food and drinking water and the second to mosquitoes which breed in stagnating water. Dentists practicing in rural areas and near urban slums are specially advised to keep alert to these differential diagnoses.

Vasanthi G et al (1994)[21] studied the serum ferritin level of adolescent girls belonging to an urban slum and rural areas.The prevalence of iron deficiency was defined as serum ferritin less than 12 micrograms/dl. 37.5% of the rural girls especially below the age of 12 years showed evidence of anemia. Iron deficiency was of much higher order in the rural girls irrespective of the menstrual status.

Verma M et al (1998)[20] assessed the prevalence of anemia in urban school children in Northern Indian state of Punjab and found that vegetarians and girls specially those who had started menstruating were at high risk for developing anemia.

Apart from hemolytic anemia caused by malaria, other causes of destruction of RBCs are sickle cell disease, thalassemia, congenital hemoglobinopathy and other rare causes.



FIGURES 2.6A and B: Showing nutritionally compromised women who complained of burning sensation of the tongue. They had features of bald tongue, depapillation and angular cheilitis. In many states in India micronutrient deficiency is seen in alarming proportions, specially in rural poor and women with multiple pregnancies (Omal PM , Bailoor DN 2004 YDC, Mangalore)

One of the rare causes to be evaluated is bone marrow depression. Here apart from RBCs other cells like WBC and Platelets also fall in number. Common reason for the bone marrow to become dysfunctional could be drugs like chloramphenicol, anti-convulsants, phenyl-butazone, cytotoxic drugs, etc. viruses; chemicals like heavy metal poisoning, benzene etc. In some cases the cause is unidentified and clinicians use the label idiopathic.

Dental Implications

Whenever general anesthesia (GA) is to be given it is important that correct level of oxygenation is achieved. Hb level of less than 10 gm per dl is considered very dangerous and a general contraindications for GA. In known vitamin B_{12} deficiency nitrous oxide should not be a option since number of complications is more.

Moeller's glossitis is a typical pattern of red lines without depapillation commonly seen in B_{12} deficiency, atrophic glossitis in which glossy smooth depapillated tongue is seen and in many patients no changes only mild burning may be observed; are all the classical tongue changes in anemia.

Candidiasis of oral mucosa is very frequently associated with all form of anemia. Angular cheilitis is also a common presentation, in most anemias in India since they are seen in nutritionally compromised patients.

Summary

Anemia is one sign that a practicing dentist can easily note due to characteristic changes in the oral cavity. He must try to see that he evaluates the cause of this anemia and refers the patient to hematologist in recalcitrant cases.

HEPATITIS

Demas PN and McClain JR (1999)[16] have mentioned about the pathophysiologic alterations that take place in the patients with hepatic disease and the precautions that need to be taken . The three main problems associated in dental surgery with such patients is –

a. Bleeding diathesis
b. Transmission of viral hepatitis
c. No drugs are to be prescribed which are metabolized in the liver mainly.

Hepatic disease could be caused by many factors only some are mentioned below-

1. **Congenital Problems**
   a. Crigler-Najjar syndrome
   b. Rh incompatibility
   c. Gilbert syndrome
2. **Hepatocellular disease**
   a. Viral hepatitis
   b. Drug-induced hepatitis
   c. Cirrhosis
3. **Obstructive hepatic disease**
   a. Carcinoma of pancreas
   b. Gallstones

Following are some of the drugs, which are contra-indicated, in hepatic dysfunction:

A. Antimicrobials—Tetracyclines, erythromycin esteolate, talampicillin
B. Antidepressants—MAO inhibitors
C. Analgesics—Aspirin, codeine, mefenamic acid, phenylbutazone, indomethacin
D. General anesthetics—Methohexitone, thiopentone, halothane.
E. Others—Anticoagulants, oral contraceptives, prednisone, lomotil, liquid paraffin etc.

[Note* this is not a comprehensive list just an indicative one.]

Infectious Hepatitis or Hepatitis A

Hepatitis A is spread by fecal oral route and is endemic in all parts of India more so in the rural regions where same pond is used for bathing and drinking water. Nausea, vomiting and severe weakness with jaundice are characteristic. Nutritional support and rest usually takes care of the patient within two weeks and by 60 days patient is totally Ok in most cases. Carrier state and conversion of this disease into chronic disease in not reported. Dental implications are not to do any disease during active disease but call the patient after two months.

Serum Hepatitis or Hepatitis B

It is a sexually transmitted viral hepatitis, which is deadly. Dentists are prone to get this disease occupationally since we all come in contact (our gloves come in contact) with the blood of the patient. Incubation is 3-6 months and malaise, jaundice, vomiting, weakness and prostration is characteristic. It is acquired through blood transfusions, sexual contact or intravenous drug abuse. Surgeons and hospital staff are at high risk due to constant contact with infected or potentially infective patients. Depending on the patients immunity the patient may return to healthy state, or may turn into chronic hepatitis (and later into

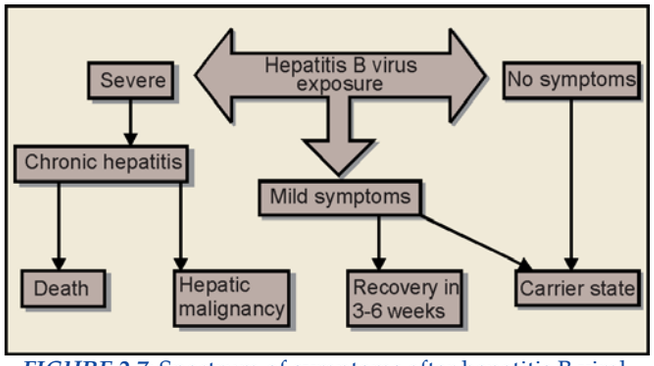


FIGURE 2.7: Spectrum of symptoms after hepatitis B viral exposure (Bailoor DN, Mahima Patil 2004)

hepatoma), or may return to apparently healthy state but be a carrier and transmit the virus actively (10-15%) and some unfortunate ones (1-2%) may face fatality (see Fig. 2.7).

Hadziyannis SJ et al (1999)[17] has recommended the antiviral Ganciclovir in the dose of 3 to 6 g in his clinical trial of 15 consecutive patients of hepatitis B. They treated them with this dose for 8 weeks and found that this can achieve sufficient suppression of HBV replication. Vaccination using recombinant DNA vaccine like Engerix, has become routine in most Indian hospitals and universities for protection of dental students and dentists against the scourge of hepatitis B.

Davies GR (1994)[18] mention that longevity of immunity is dependent upon antibody levels attained by the immunization program. Postimmunization antibody levels can be utilized as a guide to the longevity of the immunity and to customization of the vaccination dosage.

Jorge J Jr et al (1994)[19] have indicated that Oral lichen planus appears occasionally to be associated with systemic infection with hepatitis B or hepatitis C viruses.

CIRRHOSIS

Cirrhosis of liver is the end-result of long-term insult to the liver parenchyma from a variety of poisons like alcohol, viruses and drugs.

Cirrhosis chiefly affects middle aged and elderly patients. The clinical features mainly come from diminished functional capacity of liver cells and due to portal venous hypertension.

Jaundice, weight loss, loss of appetite, and in severe cases ascitis and fluid retention may be seen. Alcoholic cirrhosis is associated sometimes with sialadenosis, gastric ulceration, pancreatitis, hyper pigmentation and a sign rarely seen nowadays called Dupuytren's contracture.

Dentist, in known patients of cirrhosis, should not prescribe following drugs.

1. Tetracyclines have liver toxicity in high doses and may be avoided.
2. Erythromycin stearate may be prescribed the esteolate is harmful.
3. Halothane anesthetic is known to cause liver toxicity in middle aged, obese females.
4. Aspirin when given to children who have any form of viral infection may precipitate severe liver damage with CNS, problems. This is termed as Reye's syndrome.
5. Other newer drugs should be checked for liver toxicity in the manufacturers folders or internet sources like Medline or Medlars etc.

Liver cancer or hepatocellular carcinoma (HCC) is the fourth commonest cause of cancer deaths in the world. The condition is extremely common in Southeast Asia and Africa. Dhir and Mohandas (1998)[22] estimate that 12,750 new patients will be diagnosed to have HCC in India in the year 2001comprising of 1.6% of all incident cancers. The contamination of foods with aflatoxin and the moderately high prevalence of hepatitis B (HBV) and hepatitis C (HCV) virus-related chronic liver disease in India, has complicated the picture of the risk factors. Published data supports hepatitis B virus to be the single most important risk factor.About 80% of Indian patients with HCC have hepatitis virus-associated liver disease.

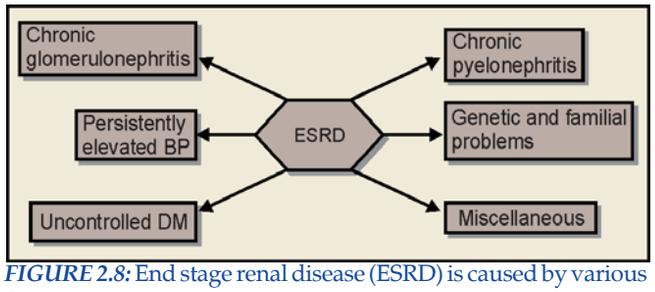


FIGURE 2.8: End stage renal disease (ESRD) is caused by various factors. Six common ones depicted above (Bailoor DN, Mukta Motwani 2004)

Renal Dysfunction Patients and Dentist

End-stage renal disease [ESRD] is the culmination of a long list of pathological processes that may damage the kidney. Commonest are shown in Figure 2.8.

Most of the ESRD patients are regularly hospitalized and undergo dialysis and or await renal transplantation where possible. Most of these patients are likely to consult hospital based dental departments. If some of them during periods of remission do come to regular dental clinics, initial past medical history and the record itself should alert the dentist. It is best to refer such patients after initial pain and infection management to hospital based set up.

Dentist must be concerned about the following:

1. Bleeding diathesis and anemia
2. Hepatitis B and other viral infection
3. Hypertension (uncontrolled)
4. Bone marrow depression
5. Drugs mainly metabolized in the kidney
6. Bone changes related to ESRD
7. Spread of infection from the oral regions.

ESRD mainly affects the platelet functioning through increased prostaglandin I, decrease in Factor III and problem with platelet aggregation. Patients are mostly on heparin so it is possible that this all complicates the underlying bleeding tendency. Best time for dental treatment is a day after dialysis but as usual as a team with the renal specialist and hematologist in hospital set up.

Immunosuppression due to treatment by steroids and other agents like azathioprine warrant that these patients be kept on bactericidal antibiotics prior to any dental manipulations.

Drugs that are excreted mainly in kidney should be replaced. Tetracycline, cephaloridine, phenacetin and phenylbutazone can cause severe damage to the weak kidney and are some of the drugs best avoided.

Bone changes related to ESRD – When patients undergo the regular hemodialysis there is increasing levels of phosphate in the blood. This leads to lowering of calcium levels and stimulates parathyroid activity. This is one of the causes of secondary hyperparathyroidism. Vitamin D metabolism is impaired by renal problems because the 1,25-DHCC (1,25 dihydrocholecalciferol) is not formed in the renal cells and this impairs the calcium absorption. This further aggravates the calcium levels.

The rennin-angiotensin mechanism results in hypertension either being initiated or aggravated by renal disease and is a factor needs to be kept in mind for drug interaction with anti-hypertensive therapy and complications of dental treatment in hypertension.

The ESRD patient is a serious patient usually hospitalized and a team approach always including a nephrologist is the best way to treat any dental complications in hospital. Such patients are unlikely to walk into a dental clinic as out-patients.

SUMMARY

This chapter tries to address a very vast area of knowledge of medically compromised dental patient. This chapter is merely indicative of different risks and when the dental surgeon is in doubt always follow a golden rule – take the phone and talk to the specialist. In all cases patients taking any medical treatment need written consent from the physician or family doctor about the fitness to receive dental treatment. All these consent notes need to be stapled to the primary dental record of the patient and entered in his computer file. If the dentist feels that after his treatment the patient might need physician's care then clear written instructions and telephonic follow-up is a good way to develop healthy rapport for total health care of the patient.

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NOTE

This chapter is not a replacement for professional dental training. Kindly verify the latest prescribing practices with your teachers and consultants prior to making real life decisions. Most values are indicative and have been checked against latest reliable sources, but the publishers and editors do not have any direct or indirect liability to the use or misuse of this prescribing information.

Prior to prescribing any medication please check that they are from ethical drug manufacturers following sound quality control practices. Follow the manufacturers directions in most prescriptions and in case of new drugs please confirm side effects, safety in children and pregnancy with the nearby-approved University hospital specialists and legitimate internet sources.

Editors

Bailoor DN, Leela Krishnaprasad,
Pai Keerthilatha, Mahima Patil

3 Dental Evaluation and Management in Pregnancy

INTRODUCTION

Many Dental surgeons in India appear reluctant to treat pregnant dental patients in their clinics due to unreasonable fear of complications. However, the importance of good oral hygiene can hardly be emphasized at this juncture in life. The following maternal changes are revised for the practitioner in order that he may rationally base his treatment keeping them in mind.

FIGURE 3.1: First trimester of pregnancy is difficult to detect and alert dental surgeon must ask proper history (Bailoor DN, Krishnaprasad L 2004)

MATERNAL CHANGES

- ***Endocrine:*** Multiple hormonal changes.
- ***Cardiovascular:*** Increase of 20 to 40% in cardiac output, tachycardia, and murmurs.
- ***Hematologic***: Increase of 30% in maternal blood volume.
- ***Respiratory:*** Increased rate of respiration.
- ***Complications:-*** Spontaneous abortions: Probability of 15% during the first trimester. Possible relationship to stress or bacteremia.
- ***Ectopic pregnancy:*** Fertilization and implantation of the fetus in a fallopian tube, resulting in abdominal pain and heavy bleeding.
- ***Pre-eclampsia*** is marked by hypertension and proteinuria in some cases. Eclampsia is characterized by malignant hypertension, seizures, and encephalopathy in some cases.
- ***Hypertension and syncope:*** Secondary to fetal compression of the inferior vena cava should be checked for.
- ***Anemia:*** Secondary to increased hematological demands.
- ***Cardiovascular diseases:*** Exacerbation of underlying disease in response to increased demand.
- ***Oral:*** Exacerbation of underlying periodontal disease. Increased risk of pyogenic granuloma, and pregnancy gingivitis.

GENERAL GUIDELINES

Take a history of the trimester and note complications of past pregnancies and blood pressure (see Fig 3.1).

First Trimester

Fetus is especially susceptible to teratogenic influence and abortion. Evans RD (1994)[4] mentions that prolonged pregnancy induced vomiting in the first trimester can cause severe chemical erosion in the palatal surface of the upper incisors. This indicates that the dentist must see such patients early on and if too much vomiting is evident patient should be encouraged to use baking soda mouthwashes to neutralize acidic content of saliva.

Second Trimester

Optimal trimester for dental care. Machuca G et al (1999)[5] have studied periodontal status of 130 pregnant women and found that the mean plaque index was high in lower socioeconomic groups and when patients lived in rural areas. This once again highlights that those patients who get early care are less likely to have plaque accumulation.

Sands TD et al[14] have mentioned that the dentist should realize the risk-benefit ratio in all therapeutic modalities in managing any pregnant dental patient. It is always best to err on the side of safety and remain conservative. The dental problem can usually be treated with the use of adequate local anesthesia and supplemental nitrous oxide in the second or third trimester. Preventive, emergency, and routine dental procedures are all suitable during various phases of a pregnancy, with some treatment modifications, initial planning and with consultation with the patient's physician or specialist.

Third Trimester

Syncope and hypertension risk are greatest secondary to fetal position. Cardiovascular demands are greatest. There is increased risk of anemia, the highest risk of eclampsia, and increased risk of hypertension.

Diaz-Romero RM et al (1998)[6] Surveyed 700 physicians in Mexico and asked them about dental referrals and found to their surprise that 53% of the doctors did not recommend that the pregnant patients seek dental care and in general the communications between doctors and dentists in relation to inter-referrals was considered poor. In India too it would be a good idea if dentists had good public relations with the local Indian medical association (IMA Chapter) and the patient's family doctor (see Fig. 3.2).

Perti C et al (2000)[3] have recommended that paracetamol is the analgesic of choice for pregnant dental patients and penicillin, cephalosporins, and erythromycin are the antibiotics recommended. It is best to weigh risk versus benefit for even these prescriptions, since in the absolute sense no chemical introduced in the pregnant woman's serum can be deemed to be safe.

Purwar MB et al (1999)[7] has interviewed 600 pregnant women in Nagpur. They have determined the frequency and severity of physical abuse during pregnancy. In majority, the abuse site was head and neck. The dentist can look for damage to teeth and facial fractures which are not easily explained by accidents or which have obvious marks of domestic violence. Whenever the dentist finds the pregnant woman or a child abused then women welfare section of relevant state government/police authorities have to be reported immediately and the report may need to be confirmed by a Government hospital/ Government surgeon in most states in India.

Aune B et al (1999)[9] state that during pregnancy changes in blood coagulation and fibrinolysis create a hypercoagulable state. The platelet count is significantly increased postpartum both after normotensive, and pre-eclamptic pregnancies. They analyzed 22 postpartum cases and found that the thrombocytosis peaks between 6-14 days, usually at a time when patients are discharged from hospital.

Atalla RK et al (2000)[10] have confirmed the above in 20 patients undergoing normal delivery and 25 cesarean delivery and found reactive thrombocytosis in all the patients with increase in platelet count which continued for about 24 days after the delivery. Dentists who have postponed the dental treatment of pregnant patients for medical reasons may like to remember this prior to initiating surgical dentistry postpartum.

Ludwig H (1999)[11] mentions that in general the risk of various bleeding disorders in pregnancy is greatly increased and the practicing dentist must take cognizance of this fact by doing preventive care and dental health education in the early part of pregnancy. When post-operative bleeding is encountered, use local methods to counter it.

An interesting finding published in the space medicine journal by Geeze DS (1998)[12] mentions that exposure to

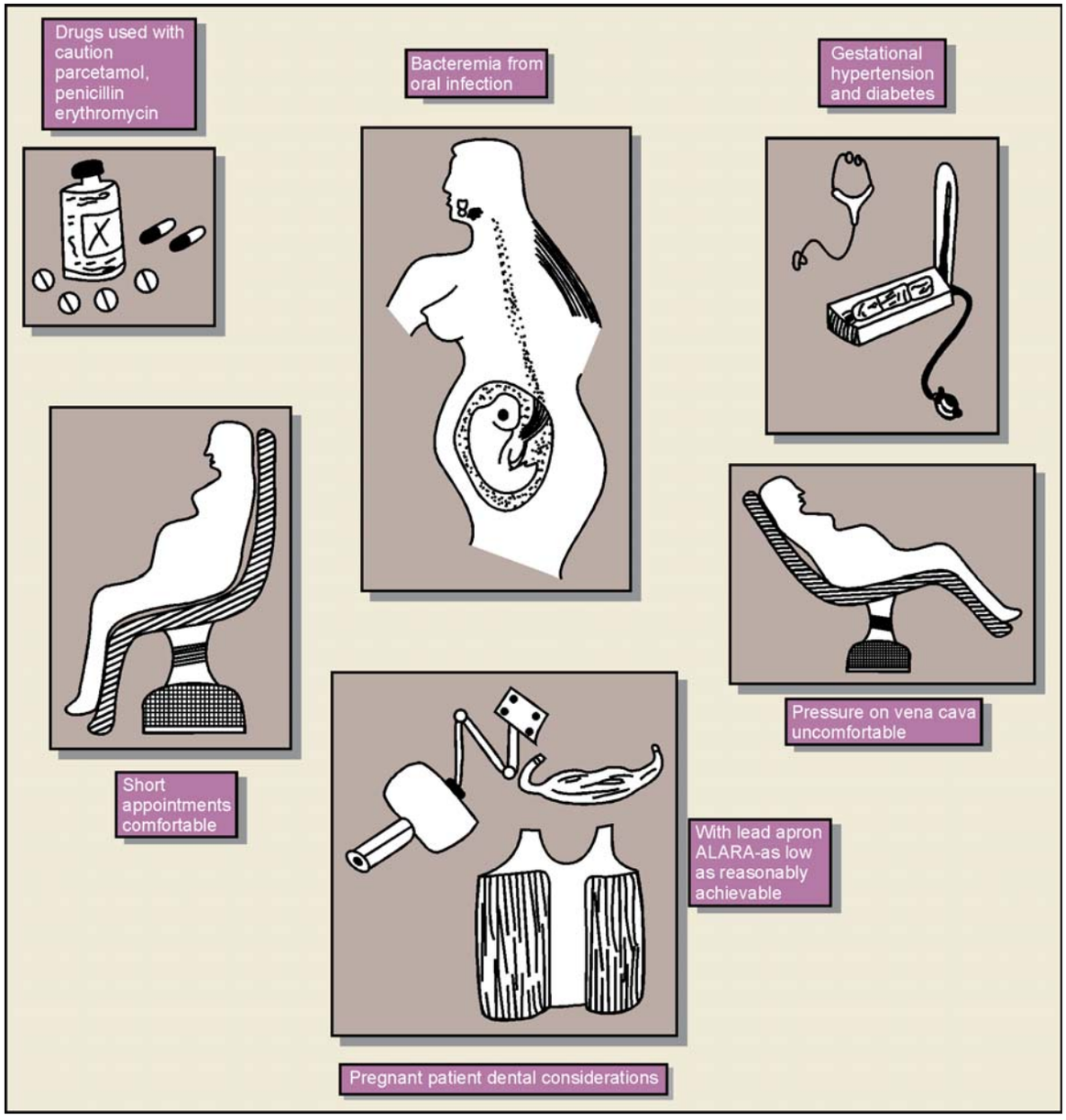


FIGURE 3.2: Pregnant patient—dental considerations (Bailoor DN, Nagesh KS 2004)

cosmic radiation is a work related risk for the flight crew, pilots, and air hostesses. When this group of aviators visit dentist, he must try to counsel them that the posting on land should be sought for the period of pregnancy since the risk of mental retardation and childhood leukemia has been documented in persons getting exposed to the increasing levels of cosmic radiation.

Romero BC[13] has mentioned that there were significant differences in the weight and gestational age of the newborns of mothers with periodontal disease (PD). They have done a study to find out whether maternal PD could be associated with the nutritional condition of newborns. From their study they concluded that there was decrease in the average newborn's weight and gestational age as

the mother's level of PD increased and also mentioned that periodontal disease (PD) in pregnant women could be a clinically significant risk factor for preterm deliveries and low birth weight.

The possible causes of caries during pregnancy are: changes in saliva and mouth flora, vomiting, neglected oral hygiene and nutritional changes. Vadiakas G et al[15] states that the increased needs of dental care of the woman during pregnancy must be emphasized, as well as her special management during the dental treatment. Treating the pregnant patient is a task of a group of specialists, which should include the dentist, too (see Figs 3.3 and 3.4).

Blagojevic D et al[16] states that pregnancy is the time when conscious approach to preventive oral care should increase. Preventive measures during pregnancy mean usage of fluorides, special dietary measures and increased oral hygiene habits. The optimal period for introducing preventive measures is the first trimester of pregnancy. Because of hormonal alterations there is an increased incidence of dental diseases. Eating habits of pregnant women may lead to frequent snacking, thereby increase the risk of caries.

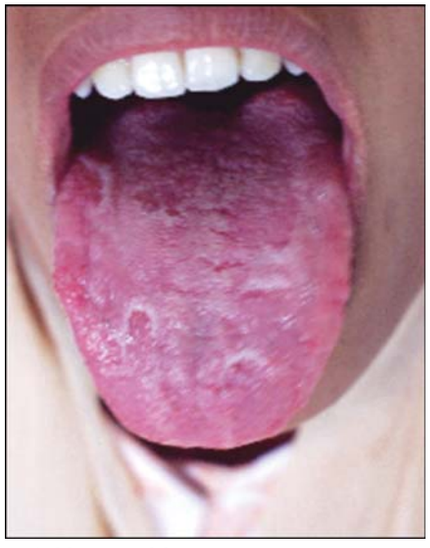
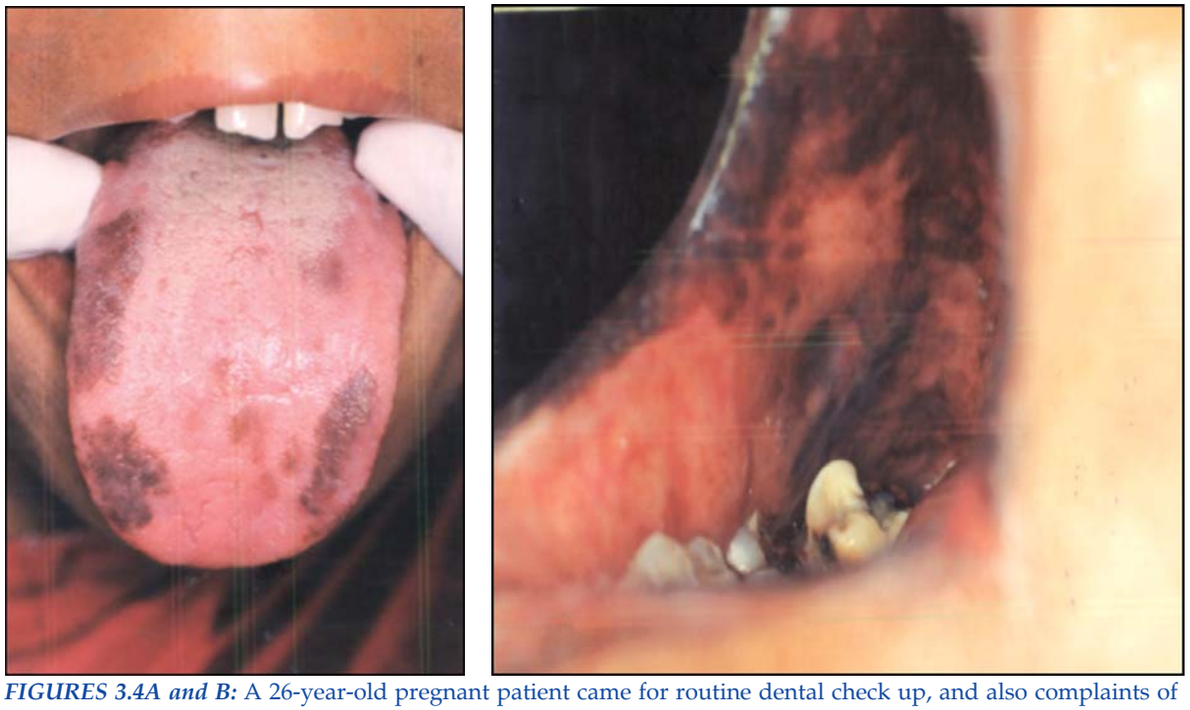


FIGURE 3.3: A 23-year-old pregnant patient came with severe burning sensation of the tongue. Nutritional factors and hormonal changes have been implicated in this case. The so called "Geographic tongue" vanished after the childbirth (Leela Krishnaprasad, 2004)

Hilgers KK et al[19] give excellent guidelines for the dental practitioners for treating adolescent pregnant patients. This becomes more relevant as the dentist will see younger and



FIGURES 3.4A and B: A 26-year-old pregnant patient came for routine dental check up, and also complaints of blackish discoloration of the tongue and buccal mucosa. Hormonal changes have been implicated in this case (Leela Krishnaprasad, Bailoor DN 2002.Yenepoya Dental College Hospital, Mangalore)

FIGURE 3.5: Showing the pregnancy granuloma in a 23-year-old female patient histopathology showed the features of capillary proliferation in a base of normal fibrous tissues interspersed with chronic inflammatory cells (Leela Krishnaprasad, Bailoor DN 2002, Yenepoya Dental College Hospital, Mangalore)

FIGURE 3.6: Showing enlarged interdental gingiva in a 28-year-old pregnant patient with a chief complaint of bleeding from the gums (Leela Krishnaprasad, Bailoor DN 2002, Yenepoya Dental College Hospital, Mangalore)

younger women presenting with pregnancy. These adolescent and very young women are not educated into the hazards of poor oral hygiene on unborn. It is upto the Gynecologists and the Dentists to form a team and initiate health programs specially for the underprivileged and the rural women.

SPECIFIC GUIDELINES

1. Preventive dental prophylaxis should be undertaken at the beginning of the second trimester and the third trimester.
2. All elective dental care should be deferred.
3. No deferrable treatment (e.g. caries control) should be completed during the second trimester.
4. Radiographs are contraindicated in all but emergency situations, when taken, lead shielding is mandatory.
5. There should be medical clearance for all drugs, including local anesthetics, analgesics and antibiotics.[2]
   a. Lidocaine, penicillin, erythromycin, and acetaminophen, (Tylenol) are generally approved.
   b. Aspirin and vasoconstrictors in local anesthesia and all drugs causing respiratory depression (e.g. narcotic analgesics) are relatively contraindicated.
   c. Diazepam (Valium), nitrous oxide, and tetracycline are absolutely contraindicated.
6. Prolonged chair time must be avoided to prevent the supine hypotension.
7. Sitting up position is best for such patients since head low position may cause pressure on the vena cava and aorta in the second trimester. Place a pillow or rolled blanket under the patient's right hip.
8. Alternately the patient is turned on the left side in the dental chair during the treatment procedure.
9. Wasylko L et al (1998)[8] clearly mention that dental treatments are best performed in the second trimester for the benefit of the fetus, and optimal comfort for the pregnant woman. Second trimester is the safest most stable time for needed treatment.
10. Elective Radiography like Bitewing technique is contraindicated in the pregnant patients. Only one IOPA or OPG according to the clinical need may be taken with proper lead shielding. In latter only 0.00015 Rads reaches the uterus during the OPG examination and so can be considered safe.[1]
11. In case of fainting, place patients on left side with legs and head elevated, oxygen and lime juice with glucose could be given and vital signs monitored. Oropharyngeal suction should be available in morning sickness.
12. More serious complications like seizures, and active vaginal bleeding or severe cramping requires

emergency care in a hospital in such cases patient should be placed on the left side, given oxygen and transported immediately to the nearest facility.

13. In cases of past history of spontaneous abortions please take a written consent from her obstetrician; otherwise an unrelated dental treatment may have to take blame for the abortion at a later date.
14. Multiple studies suggest that patients with periodontal disease appear to be linked two times more with risk of cardiovascular disease and seven times more likely to bear low birth weight infants. Paquette DW[17] (see Figs 3.5 and 3.6).
15. Koren G et al[18] indicates that counseling the women about the teratogenic risks of over the counter medications would play an important role in preventing birth defects. To date drugs like tetracycline, doxycycline and metronidazole have been implicated in problems as small as discoloration of teeth to neural birth defects. Carbamazepine, isotretinoin , phenytoin and valproic acid have been established as teratogens in various studies around the world.
16. In India, iron deficiency, vitamin A deficiency, and iodine deficiency disorder are of greatest public health significance. In addition, subclinical zinc deficiency, fluorosis, and fluoride-deficient dental caries are important areas of concern. The National Pilot Program on Control of Micronutrient Malnutrition was launched in 1995 and Chakravarty I and Sinha RK[20] reported a high incidence of anemia, night blindness fluoride deficient dental caries in many parts of Assam, Bihar, Jharkhand , Orissa , West Bengal and Tripura.

SUMMARY

Radiation exposures in women have been associated with low birth weight offspring. It could be hypothesized that hypothalamus-pituitary-thyroid axis may be affected and thereby indirectly birth weight, or that the radiation may directly affect the reproductive organs. Hujoel PP[21] state in their study that dental radiography during pregnancy is associated with low birth weight. Treatment in the pregnant women requires an understanding of the balance between maintenance of her oral health and the risk to the fetus.

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Bailoor DN, Nagesh KS

4 Temporomandibular Joint Diseases

INTRODUCTION

Throughout history of Medical treatment the disorders of the Temporomandibular Joint (TMJ) have been difficult to treat. Vast amount of data regarding the basic anatomy and the psychophysiology of the TMJ is available now, what probably is lacking is its correct application. TMJ is functionally the most important joint, because it helps in masticating, talking and even kissing. The disorders affecting the TMJ are collectively referred to as Temporomandibular Disorders or TMD in the following discussion.

ANATOMY AND PHYSIOLOGY OF THE TMJ

TMJ is unique because this ginglymo-diarthrodial joint has capability of hinge and sliding movement. This joint is actually two anatomically separate joints, right and left, functioning as one in a balanced manner. A single U shaped bone articulating with base of the skull at its two ends (Refer Fig. 4.1).

It constituents are the bony components, (mandibular condyle and glenoid fossa of the temporal bone), and the articulating disc (fibrocartilaginous elements sandwiching the bony components). Both of these are cocooned in synovial membrane surrounded by a capsular ligament. The mandible is suspended by select group of masticatory muscles, ligaments and the vertical stop is provided by the occlusion.

Neurovascular Supply

The main blood supply is from the superficial temporal and emissaries from internal maxillary artery. The nerve supply is from the Cranial Nerves VII, IX, X and XI and Cervical Nerves C2 and C3. Branch of auriculotemporal nerve supplies the sensory innervations to the TMJ.

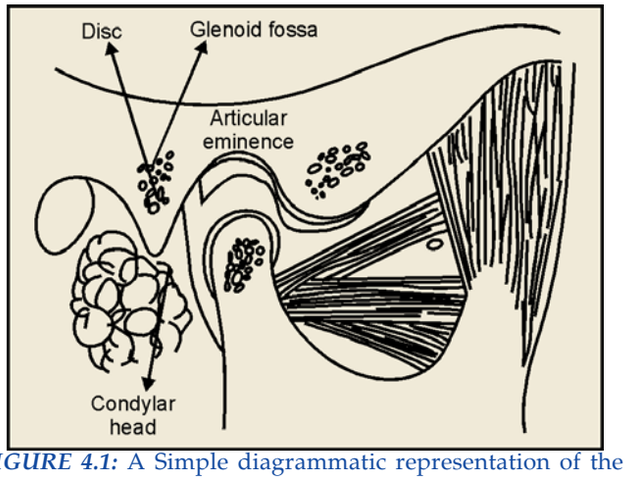


FIGURE 4.1: A Simple diagrammatic representation of the Temporomandibular Joint of the human body (Bailoor DN, Prasanna K 2004 Yenepoya Dental College, Mangalore)

Occlusion

In lay terms, occlusion means coming together of upper and lower teeth in functional and non-functional actions. This mechanical concept is now replaced with physiological occlusion, which is defined as an occlusion,

in which; functional equilibrium or state of homeostasis exists between all the tissues of masticatory system. Any disturbance in the balance results in the Kinesiological problems. In fact, the term ***kinesiology***[21] denotes a dynamic neuromuscular homeostasis and the various factors that will go into its stability and maintenance.

SYMPTOMS AND SIGNS OF TMD

Following symptoms and signs should alert the clinician to look for TMJ pathology

1. Pain in the pre-auricular region—Acute or Long standing
2. Joint noises— clicking and popping, grating, etc. related to opening or closing of joint
3. Restriction in opening, painful opening or jerky opening
4. Deviation in opening with or without restriction
5. Tenderness over following regions—the pre-auricular region, muscles of mastication, cervical region, upper back pain
6. Unilateral or bilateral headache originating from that focus
7. Swelling or redness in the TMJ region, unilaterally or bilaterally
8. Unexplained ear pain

RISK FACTORS FOR TMD

1. Emotional distress and Depression
2. Macro-trauma—blow or accidents causing hemarthrosis or fracture
3. Micro-trauma—long dental procedures, hyper extension associated with neck trauma, bruxism and clenching
4. Instability of the physiological occlusion— due to extraction of posterior teeth, high dental restorations, improperly planned dental bridges, ongoing orthodontic treatment, etc. Unbalanced Kinesiological pressures.[21]
5. Infections from contiguous areas like middle ear infection, mastoiditis, etc.
6. Systemic disorders like Rheumatic disease, Psoriasis, Gout, Musculoskeletal disorders etc.
7. Laxity of ligaments

CLASSIFICATION OF TMJ DISEASE

TMD—Temporomandibular Joint disorders

They are basically classified on 3 main Axis (I-organic, II- psychosocial, III- combination)

Usually chronic pain related disorders of the TMJ fall under Axis II or III.

I. ***Axis i*** –
   i. Organic
      1. Condylar
         a. Congenital
            i. Aplasia
            ii. Hypoplasia
            iii. Hyperplasia
            iv. Dysplasia
         b. Acquired
            i. Tumors; Benign and Malignant
            ii. Fractures—Hemarthrosis—Fibrous—Bony Ankylosis
   ii. Arthrogenous
      1. Functional
         a. Hypermobility, Subluxation, Dislocation
         b. Arthrogenous-Architectural
         c. Internal derangement with/without reduction
      2. Inflammatory
         a. Synovitis/Capsulitis
         b. Arthritis—Septic, rheumatoid, gout, psoriasis
         c. Polyarthritis
      3. Non-inflammatory
         a. Osteoarthrosis
         b. Osteoarthritis, and
         c. Ankylosis
            i. Primary/Secondary
   iii. Myogenous
      1. Myositis
      2. Myospasm
      3. MPDS
      4. Contracture
      5. Neoplasia

II. *Axis ii—Psychosocial Disorders*
   i. Depression
   ii. Somatization
   iii. Personality Disorders

III. *Combination*

Condylar Problems—Congenital (Aplasia, Hypoplasia, Hyperplasia, Dysplasia)

Developmental Problems

They occur due to either genetic or environmental factors.

- Undergrowth of the condyle (Hypoplasia)
- Overgrowth of the condyle (Hyperplasia)
- Lack of condyles (aplasia) this is extremely rare.
- Bifid condyle (see Fig. 4.2)

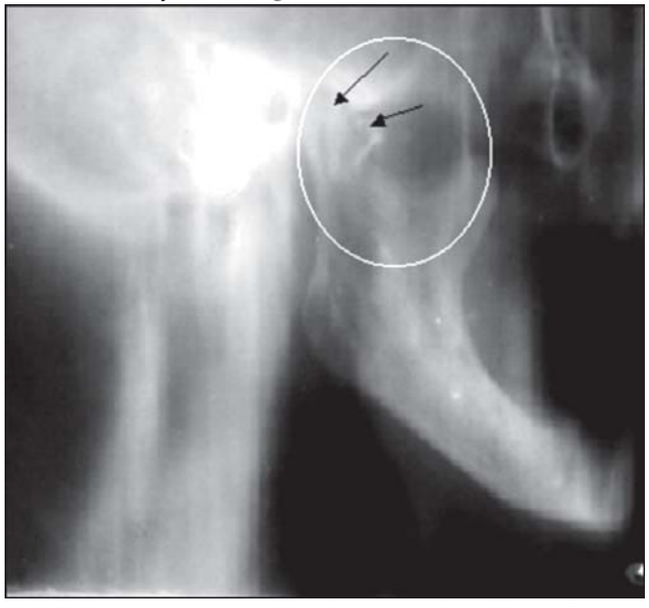


FIGURE 4.2: A sixty year old male came with problem of grating noise of the right side. Since last several years. Routine radiography revealed unique condylar. Architecture of two condylar heads. A tentative diagnosis of bifid condyle was made. Oral rehabilitation with removable complete prosthesis, warm compresses and use of Disprin® 400 mg made the patient comfortable (Courtesy of Iyengar A, Nagesh KS 2004 Department of Oral Medicine Radiology, RV Dental College, JP Nagar, Bangalore)

Condylar Problems (Acquired—Tumors, Fractures, Bony ankylosis)

Malignancies may develop in any component of the joint.

- Commonest benign tumors being osteoma, osteochondroma, chondroma chondroblastoma, fibromyxoma and benign giant cell lesion.
- Malignancies chondrosarcoma, metastatic tumors etc. are reported in an excellent report by Thoma[15] and are fortuitously rare.

Fractures of the Condylar, Subcondylar Region

History of trauma and pain and swelling over the involved condyle would obviously make the diagnosis of the fractures of this area simple. Especially with the advent of routine OPG available at all the good diagnostic centers. Its treatment, however, lies mainly in the surgeons realm. A point to be noted is that most of the workers today believe that closed reduction is the best way to treat the uncomplicated subcondylar fractures[13] (see Fig. 4.3).

Ankylosis

It is a condition in which fibrous or bony tissue replaces the joint space due to pathologic and overzealous repair mechanisms.

It is classified as * bony/fibrous * unilateral/bilateral * complete/incomplete. The various workers in the field for this incapacitating joint condition have used all these adjectives. Straith and Lewis enumerate etiologies as follows:[21]

*Abnormal Intrauterine development * Birth injuries * Trauma to the chin * Malunion of condylar fracture * Injuries to the malar region * Zygomatic compound fracture * Loss of tissue with scarring * Congenital syphilis *Primary/secondary inflammation of the joint *Hematogenous infection * Metastatic malignancies * Radiation therapy effect.

Topazian[8] reviewed 229 cases of ankylosis and found that 49 percent were as result of joint inflammation, 31 percent were trauma related 20% were idiopathic.

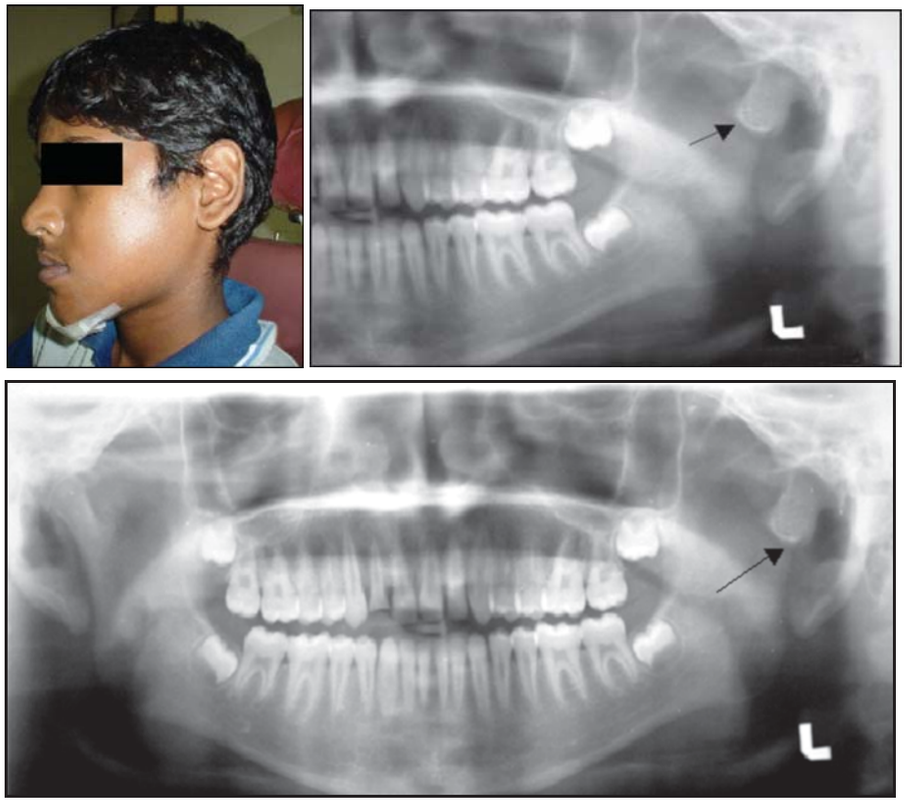
The radiographs and the history of any of the above mentioned etiologies would alert us to the possibility of surgical intervention. Surgery is definitely indicated when the ankylosis is bony but Fibrous ankylosis may be treated by the functional methods, active physiotherapy, and anti-inflammatory drugs (see Fig. 4.7).

Trauma to the TMJ—Micro and Macro Trauma

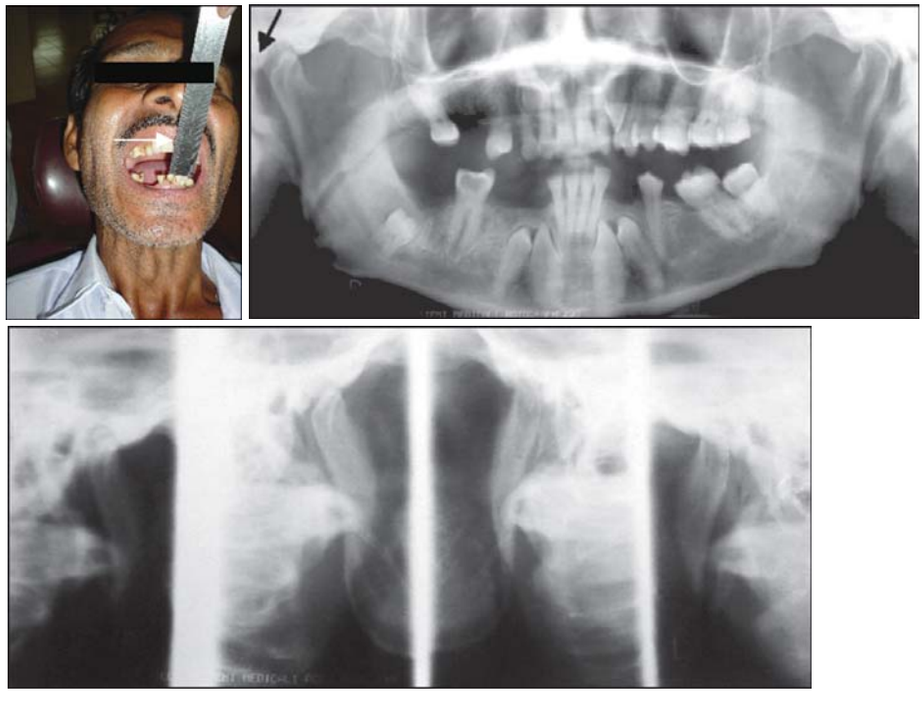
Temporomandibular joint may undergo two types of trauma—

a. Microtrauma
b. Macrotrauma

Microtrauma occurs when there is persistent low grade overloading of the TMJ due to increased stresses (see Fig. 4.4). Seen in frustrated people who suffer from bruxism,



FIGURES 4.3A to C: Showing extra articular fracture of the left condyle in a 14 year-old boy who fell from his bicycle. Chin sustained minor injury and the condyle was #. He was treated conservatively with rest and active mobilization after four weeks (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)



FIGURES 4.4A to C: 48 year old male patient came with CC of the restricted mouth opening. Following traumatic extraction, one month back. Progressive reduction in the opening was reported. OPG and sectional views revealed haziness in the right TMJ region and a diagnosis of Hemarthrosis of the joint with secondary fibrous ankylosis was made. Treatment was combination of Diclofenac sodium 50 mg once day with serratiopeptidase enzyme combination (Tolpa D®) followed with Tolpa® additionally three times a day together with hot fomentation and jaw exercises for ten days, gave uniform relief (Nillofer S, Prasanna K, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

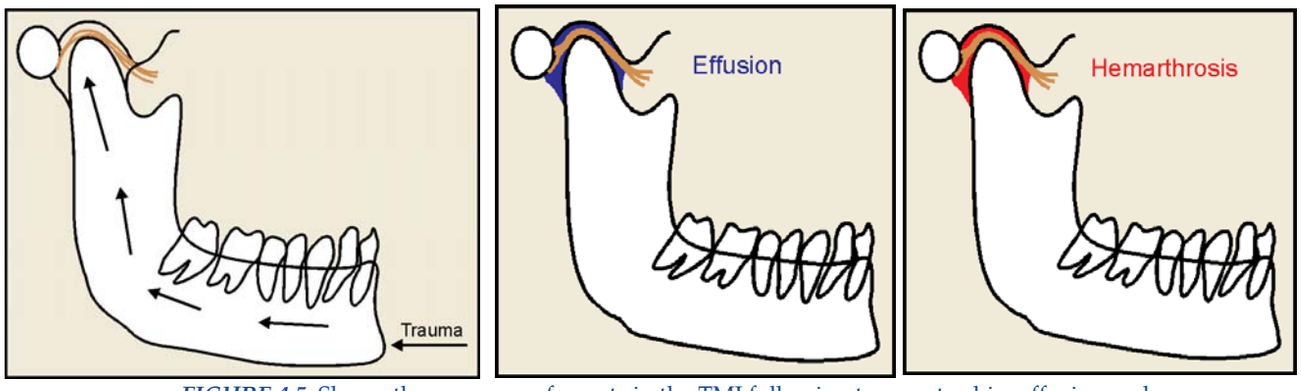


FIGURE 4.5: Shows the sequence of events in the TMJ following trauma to chin, effusion and hemarthrosis are two stages shown here (Bailoor DN, Nagesh KS 2004)

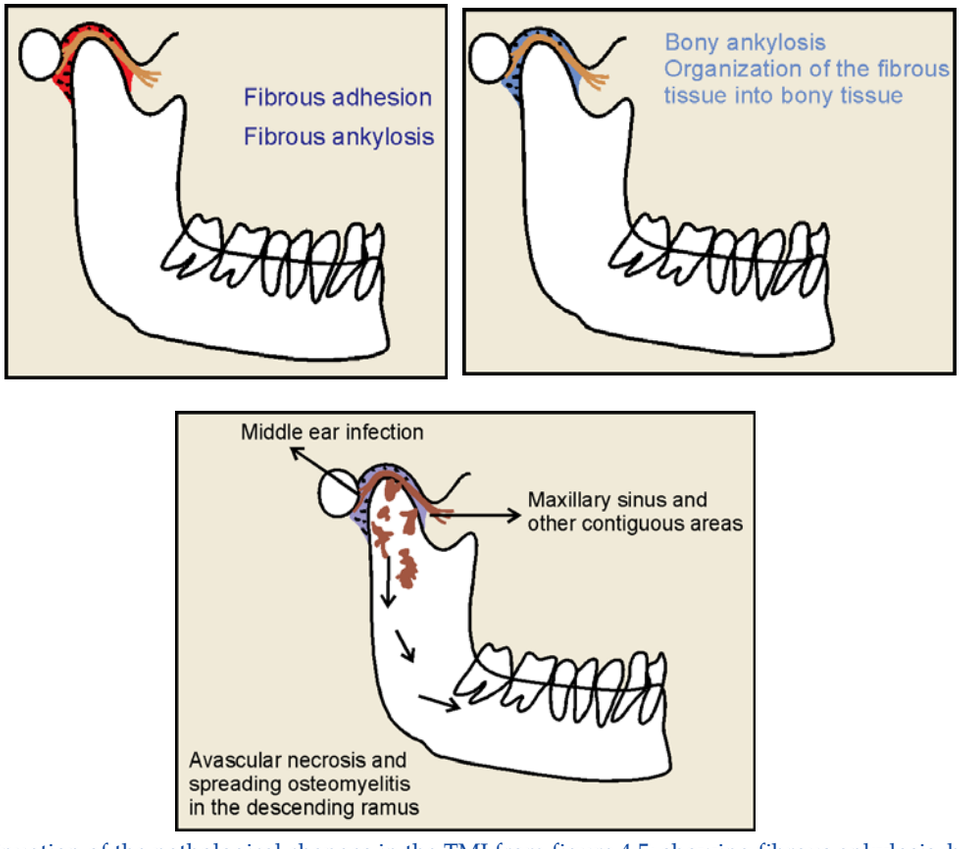


FIGURE 4.6: Continuation of the pathological changes in the TMJ from figure 4.5; showing fibrous ankylosis, bony ankylosis and avascular necrosis and its spread to contiguous areas (Bailoor DN, Nagesh KS 2004)

clenching and other parafunctional habits or in musicians who play wind instruments, violin, trumpet, etc. (see Figs 4.5 and 4.6).

Macrotrauma could be of two types again

1. Due to fall, road traffic accidents or fights etc.
2. Due to traumatic extraction, excessive pressure is caused or required for the extraction and hence, may also be termed as iatrogenic.

Both the kind of traumas may cause the TMJ to undergo gradual changes, which may have gradual but far

FIGURE 4.7: Showing a 31 year old male with post ankylosis surgery performed. Case from OPD of Yenepoya Dental College and Hospital, Mangalore (BH Sripathi Rao, Gunachandra Rai 2003)

reaching effects. The diagrams below is attempted to hypothesize these changes and how they may cause the clinical problems to the patients.

First stage is formation of inflammatory exudates termed as the effusion and it may cause mild clicking and pain. The next stage is the formation of the blood clot into the joint and may termed as the hemarthrosis. This may organize and give rise to the fibrous adhesions and later fibrous ankylosis. If the condition is still neglected by the patient and remains untreated then it gradually progresses into bony ankylosis. This leads to avascular necrosis of the adjoining areas and sets the stage for the infection to set in into the bone and contiguous tissues. Middle ear infection, maxillary sinusitis, and descending osteomyelitis of the ramus could all be possible sequels to this focus of infection.

Subluxation/Dislocation

Subluxation is a form of hypermobility, either due to laxity of the ligaments or due to the change in the fossa and eminence architecture. There is a self-reducing dislocation of the one or both TMJs. *Dislocation* on the other hand is a description of the condition precipitated by force or trauma where the patient himself cannot reduce the condylar position which has moved out of the glenoid fossa, and the mouth remains in an open position when he sees the doctor, or the mandible is open with marked deviation to the opposite side to the joint affected.

Treatment includes using muscle relaxants and guiding the mandible by an inferior and posterior push followed by immobilization of the jaw by strap bandage around the head and counseling not to open wide for a period of one to two weeks.

Internal Derangements

This condition is characterized by the disc in a abnormal location in relation to the other components of the joint. Usually it is displaced anteromedially and there is loss of normal joint function. Diagnosis is best established by Double contrast Arthrography[2] and by CT examination[6] usually required to confirm the anterior displacement of the disc[1] (see Fig. 4.8).

The only clinical finding may be clicking or popping of the joint and that may be faint to loud. Silver and associates[14] in their study mention that 90% of these occur in females. In some conditions patients may report with inability to open the mouth. Internal derangements may be an important factor in the cause of Osteoarthritis of the TMJ .[12]

Shafer et al[13] have used the term 'Injuries of the Articular Disc' and mentioned the commonest cause to be Malocclusion. Trauma and Rheumatoid arthritis have been mentioned as the precipitating factors.

Immobilization of the jaws to give rest, correction of Malocclusion and surgical treatments like menisectomy has been proved beneficial.

Hase M et al[20] has evaluated 75 patients in his arthroscopic examination study and found that progressive maturation of adhesions in the patients were seen in long standing symptoms. He has opined that when adhesions have matured arthroscopic interventions may be helpful in the reduction of the time required for opening.

Myofacial Pain Dysfunction Syndrome (MPDS)

This disorder is characterized by patient coming with a chief complaint of dull to sharp preauricular pain, which is more in the morning. Laskin[4] has given specific criteria to diagnose this common disorder of the TMJ seen in the young women (see Fig. 4.9).

• Unilateral dull pain in the ear or preauricular region commonly worse on awakening • Tenderness of one or more muscles of mastication on palpation • Clicking or popping noise in the TMJ • Limitation or deviation of mandible on opening • No evidence of organic changes on radiographic or biochemical testing in the initial stages.

Once the diagnosis of the MPDS is established then the clinician judges the severity of the symptoms and chooses his treatment from among the following options. • No treatment only counseling • Physiotherapy Hot/cold • Splint therapy • Analgesics/anti-inflammatory medications • Antidepressants/Anxiolytics • Jaw Exercises • Premature contact removal/Occlusal rehabilitation • Hypnosis • Meditation • Acupuncture • Psychotherapy • TENS

Normally the clinician customizes the treatment according to the needs of the individual. The Oral Physician, the Clinical Psychologist, Prosthodontist and sometimes even the psychiatrist has to intervene in the

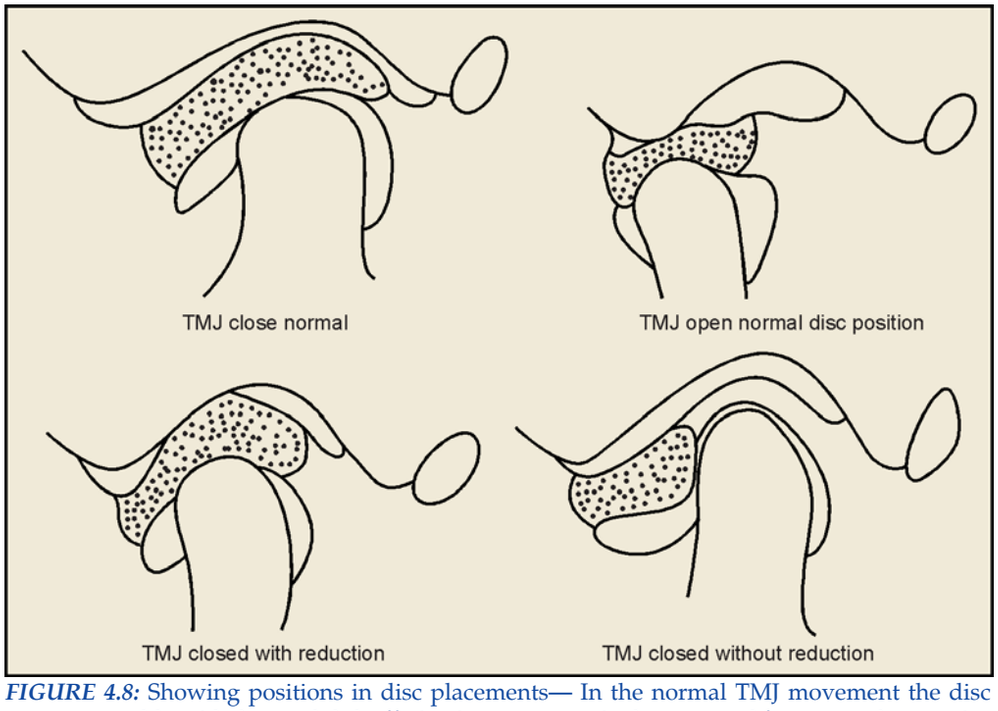


FIGURE 4.8: Showing positions in disc placements— In the normal TMJ movement the disc moves smoothly with the condyle buffering its movement in the temporal fossa upto the anterior eminence. The Disc snaps back to position in the closed with reduction problems and in closed without reduction the disc is herniated outside the joint and in extremely painful position (Bailoor DN, Nillofer Shabnam, Prasanna Kumar 2004)

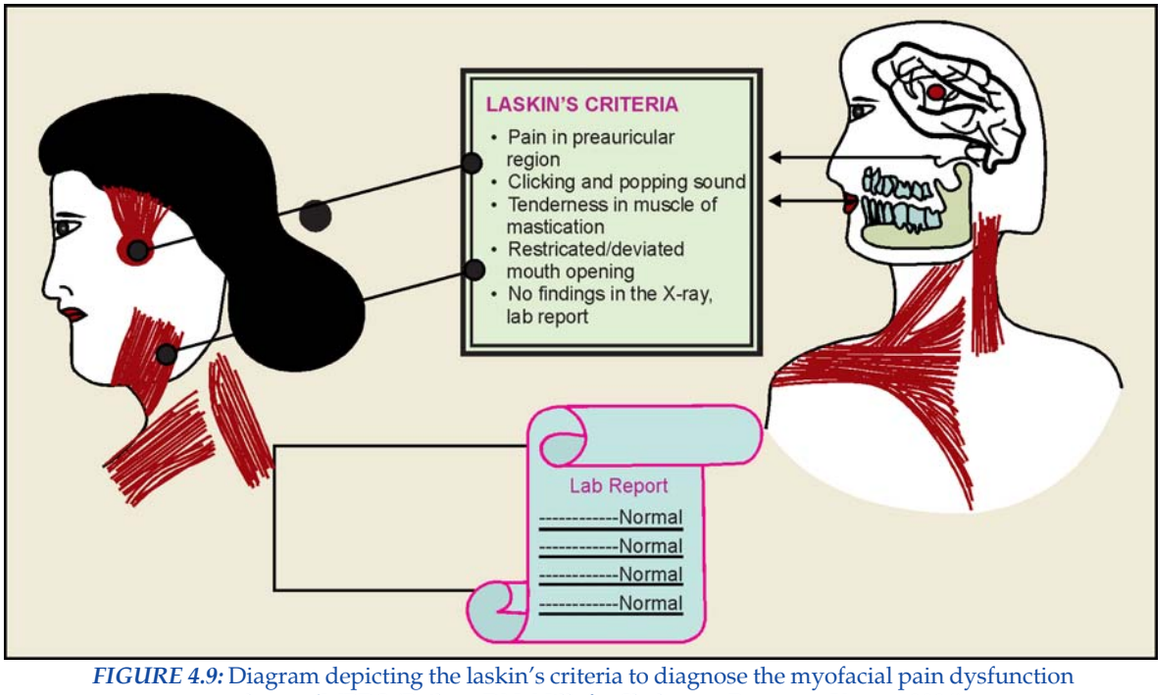


FIGURE 4.9: Diagram depicting the laskin's criteria to diagnose the myofacial pain dysfunction syndrome/MPDS (Bailoor DN, Nillofer Shabnam, Prasanna Kumar 2004)

Table 4.1: MPDS Treatment—Differentiation into mild, moderate, severe (Bailoor DN, Nages KS 2004)

Symptom/finding	*Mild*	*Moderate*	*Severe*
Clicking/popping	+	+ +	+ + +
Primary complaint	No	Yes	Yes
Muscle tenderness	One or two	All	All masticat
Occlusion problem	Nil	+	+ +
Psychosocial indicators	Non	+ +	+ + + +
Radiographic	No finding	None	Secondary osteoarthritis
Had questionnaire	Nil	Mild anxiety	Severe anxiety, mild depression
Multi-disciplinary	Not required	Must	must explore surgical option

interdisciplinary treatment modals designed for the patient (refer Table 4.1).

Bailoor et al[9] have clearly demonstrated the psychological implications in MPDS and how the screening of the patients can be done with special questionnaires in their Manipal study. It must be emphasized that such treatment involving differing specialities is best attempted in a medical or dental teaching centers.

De Laat A[18] has mentioned research concerning the etiology of MPDS is now focussing more on muscle related problems refer to more generalized disorders such as fibromyalgia. The role of occlusal and articular factors has been brought down indicating a minor contribution. In general there is a development towards constitutional and systemic factors, more than local influences.

De Boever JA and De Boever AM[19] also agree that the role of occlusal factors in the aetiology of MPDS has been overestimated in the past.

The role of Occlusal therapy should be aimed at restoring function. In the initial phase of treatment an Occlusal splint, counseling, physiotherapy and occasionally NSAIDs, leads to relieve pain and reduction of dysfunction in most patients. A repositioning splint in cases of anterior disc dislocation is no longer recommended. Selective grinding can be done in "occlusally sensitive" patients with pain or dysfunction of muscular origin.

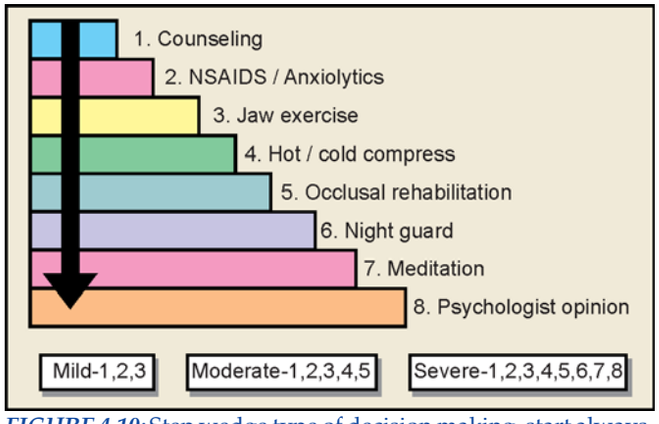


FIGURE 4.10: Step wedge type of decision making, start always with 1,2 and 3 and then proceed (Bailoor DN, Nagesh KS 2004)

McNamara JA Jr et al[16] studied the relationship between orthodontic treatment and Temporomandibular disorders (TMD) They analyzed 21 publications of studies related to the orthodontic—TMD interface and made the conclusion that orthodontic treatment was neither the cause nor could it prevent the TMD from occurring.

Korszun A et al[17] reported that 42 percent of the patients with CFS (chronic fatigue syndrome) and FM (fibromyalgia) also were treated for TMD. Amongst these patients 46 percent had histories of irritable bowel syndrome and 42 percent of premenstrual syndrome. Despite these correlations in the study 75 percent of the cases were treated purely with bite splints and only for TMD (Temporomandibular disorders). The dysregulation of the hypothalamic-pituitary-adrenal stress hormone axis in predisposed individuals appears to be the principal cause. A multidisciplinary clinical approach to temporomandibular disorders would improve diagnosis and treatment outcomes for this group of patients (see Fig. 4.10).

Lobbezoo F et al[21] have described the importance of Oral Kinesiology to the diagnosis of TMD and mention that added focus on Bruxism, Clenching, and overall the dynamic aspects of TMJ problems will result in better patient care. Netherlands is probably the first country to start the postgraduate training in this multidisciplinary knowledge area called Kinesiology.

Infective Process of the TMJ

Infective or septic arthritis[5] is usually a result of direct

spread of organisms from contiguous anatomic structures like Mastoid process, the tympanic cavity or via the blood from a distant nidus. Hematogenous spread of Gonococci or the direct spread from middle ear infection. Major symptoms and signs are:

- A sudden onset excruciating pain on opening and closing the mouth.
- A redness and tenderness over the joint area
- A inability to place the teeth in occlusion
- A large tender cervical lymphnodes
- This frequently leads to the asymmetry of the joint and ankylosis if the growth centers are involved.

Treatment

Surgical drainage from the affected contiguous anatomical part. Antibiotics parenterally may be required for periods ranging from 5 to 11 days combined with analgesics and active physiotherapy.

Rheumatoid Arthritis of the TMJ

This is the Granulomatous involvement of the synovial membrane, which is attributed to an autoimmune reaction. This spreads to the articular surface of the condyle. It occurs more frequently around 50 years of age, more in females. Radiographic and clinical signs are always diagnosed first in the small joints of the hands and feet much before the TMJ gets involved. Symptoms include bilateral stiffness crepitus tenderness, and swelling over the joint. Mostly it results in Fibrous ankylosis as a complication. Marie-Strumpell type of Rheumatoid arthritis frequently involves the Sacro-Iliac, Hip, spine and the TMJ.

Juvenile Rheumatoid Arthritis (Still's Disease)

It is a chronic synovitis with or without extra articular manifestations. Polyarthritis of both the small and large joints including cervical spine, neck pain. Radiographic features are characteristic. Mean age 5 years. One or both TMJ are involved in 40% of the patients. Two percent cases show characteristic Micrognathia. In both the above disorders Radiography shows hypoplasia/ankylosis and characteristic Antigonial notching and biochemical tests of increased ASO (Anti Streptolysin ®) titer, and Raised ESR and Eosinophilia would give important pointers.

Treatment

Good coordination with the family physician is the key to treatment of rheumatic TMJ problems. Normally Bactericidal antibiotics, Anti-rheumatic drugs like Aspirin and NSAIDs like Ibuprofen are indicated. In case of ankylosis setting in surgical intervention and post-surgical physiotherapy are indicated.

Osteoarthritic Changes in the TMJ

The wear and tear changes or the aging changes are termed as the osteoarthritic changes in the TMJ. It is classified into primary and secondary.

- Primary is when the aging process takes its toll and joint is unable to function within its normal limits. It is a degenerative disorder of the TMJ. Primary occurs in 50% of those older than 40 years and in 85% of persons older than 70 years[6] (see Fig. 4.11).
- Secondary when it occurs in individuals in the second and third decade of life when there are unreasonable stresses on the TMJ. Bruxism, MPDS and playing of some of the musical instruments are the commonest causes.

Initial destruction of the soft tissue components of the joint and subsequent erosion and hypertrophic changes in the bone, result in the wearing out of the meniscal disc and irregularities in the condylar surface, giving rise to characteristic grating or crepitus, discomfort/pain and limitation of jaw movement. Radiographic features of the later stages are irregularities seen in the condylar surfaces, Arthroliths or focal calcifications in the joint, Focal radiolucencies in the subsurface area termed as Cysts of Elyes.[7]

Occlusal rehabilitation with prosthodontic appliances are known to alleviate the pain and discomfort if not completely reverse the pathological changes.[7] Analgesics like Mefanamic acid; Aspirin and Ibuprofen appear to give uniform symptomatic relief.

Gout

Arthritis related to the metabolic disturbances of gout initially involves the joint capsule and in later stages progresses to degeneration of subcondral bone. Gouty TMJ

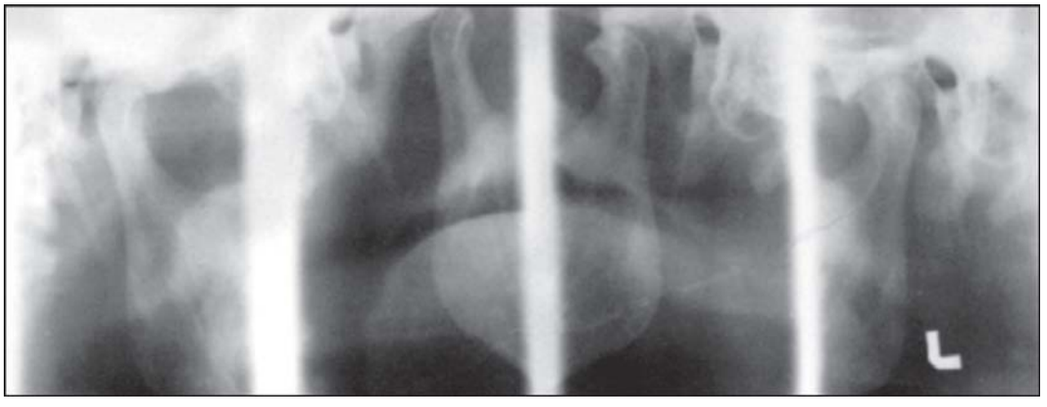


FIGURE 4.11: Sixty-three-year-old male came with CC of severe pain and crepitus of the left TMJ, radiographically pointed cone shaped condyle was noted radiographically and this "lipping" is usually seen in osteoarthritis cases. This patient also had severe tenderness in the left knee and ankle joint and on medication since last six years for OA of the left leg. (Omal PM, Beena K, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

arthritis appears to be very rare. Serum Uric acid estimation is the crucial diagnostic test. The crystals appear to precipitate in and around the joint tissues. Sub-clinical Gout has been mentioned by Bell WE[10] in which serum Uric acid level may run no higher than 6mg/dl. Filipinos, Chinese and Japanese are said to be more susceptible to this condition.

Psoriatic Arthritis

Is a rare cause of arthritis. Diagnosis is based on patients history of psoriasis and negative rheumatoid factor. Symptoms are very similar to those seen in Rheumatoid arthritis. Pain is unilateral. Diathermy, Physical therapy, exercise and Salicylates are suggested lines of treatment by Sanders and Halliday.[11] Use of Immunosuppressive drugs like Methotrexate in severe cases has also been advocated.

Diagnostic Methods

Detailed history, through clinical examination of all the cranial nerves and both the TMJs, alerts the clinician to the requirement of the various diagnostic aids necessary. Routine Radiography, CT scanning[3] and MRI constitute aids used by specialists to determine whether medical or surgical treatment is indicated in the TMJ disease.

SUMMARY

It is evident from the brief discussion of the various disorders that TMJ disease diagnosis and treatment provides a veritable challenge to the practicing Dental Surgeon as well as Dental Specialists who undertake this difficult task.

The need to use interdisciplinary treatment plans can hardly be emphasized. This complex subject and its application to the TMJ treatment is still in a state of continuous flux with new treatment modalities emerging every day. In diagnostic procedures, it is advised not to involve any invasive procedures in the patient until we have measured the risk versus the benefit, cost and utility of the diagnostic modality. This is specially so because with the advent of MR imaging the role of arthroscopy is likely to be very narrow. One of the important principle in treatment is when in doubt, do conservative treatment.

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5 Maxillary Sinus and its Dental Implications

INTRODUCTION

The maxillary sinus is the largest of the paranasal sinuses and the posterior maxillary teeth apices lie in close proximity to its floor. This means that the periapical infection can be carried to the maxillary sinus and in turn pathology of the sinus can penetrate through the roof of the oral cavity and appear as a diagnostic perplexity to the dental surgeon (see Fig. 5.1).

Maxillary sinus or the *Antrum of Highmore* is a natural cavity in the body of the maxillary bone covered with a pseudostratified squamous epithelium. It communicates with nasal fossae through the half moon shaped hiatus semilunaris. The mucociliary mechanism serves to remove the particulate matter and bacteria.

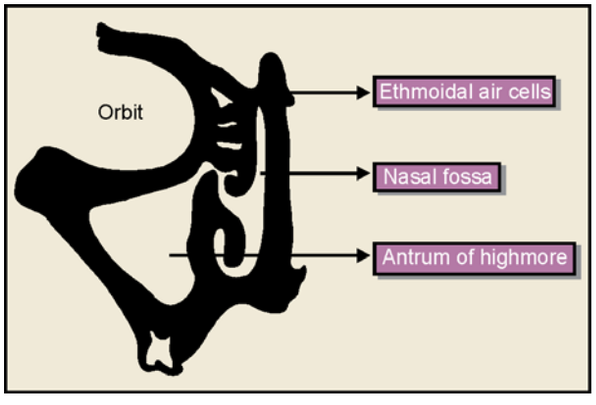

FIGURE 5.1: Coronal section figure depicting the close relationship between the floor of the maxillary sinus and the molar teeth (Prasanna K, Bailoor DN 2004, Yenepoya Dental College and Hospital, Mangalore)

The function of the maxillary sinus appears to be to lighten the skull bones without compromising the strength. It imparts resonance to the voice, and regulates temperature of the inhaled air. The protective mechanism of the nasociliary mechanism appears, however to be purely speculative.

RADIOGRAPHY OF THE MAXILLARY SINUS

The radiographic visualization of the maxillary sinus is best done by Waters Waldron view or now termed as the 15° Occipitomental radiograph. Orthopantomograph appears as the next best logical choice of the floor and lateral wall of the sinus is visualized correctly. In fact both these views complement each other and in our department, often both views are recommended to complete the radiological evaluation of the sinuses. Chronic maxillary sinusitis is depicted by opacification or haziness of the affected side. Many a time only hint is the radiopaque thickening of all the walls. Pus level and fluid level is represented by half opacification of the sinus. Many a time a dome shaped radio-opacity is seen suggesting a mucosal cyst.

Harrison DFN[1] advocates the introduction of 50% Hytrast radiopaque solution through the inferior meatal

trocar to outline intra-antral cysts. The good old IOPA technique helps in outlining the oro-antral communication if a gutta percha point is inserted into it and then the area is radiographed.

Zizmor and Noyek[2] have listed beautifully the list of radiographic appearances that may appear as the result of maxillary sinus disease.

- Generalized sinus opacification
- Generalized mucosal thickening
- Discrete soft tissue mass
- Fluid level
- Localized calcification
- Teeth or roots in the maxillary sinus
- Foreign bodies
- Alterations in the bone walls

(i) Decalcification (ii) Hyperostosis (iii) Expansion of bone walls (iv) Decreased volume (v) Effacement (vi) Discontinuity

Transillumination of the maxillary sinus yields useful additional information. In a darkened room, fiber optic light is placed in the mouth and directed towards the palate. Any prosthesis should be removed prior to this testing. Uniform crescents of light will be observed bilaterally just above the infraorbital rims at the position of the eyelids and the pupils will both glow if both sinuses are healthy.[3] If any cystic or solid mass is there in the sinus then that side the illumination shall not occur.

Recent Techniques

CT scan provides the most accurate and non-invasive technique for the evaluation of the sinus disease and the contiguous areas. Palacios and Valvassore.[4]

Ultrasound technique is useful in both the assessment of the lymph nodes in this area as well as the differentiation between tumors, fluid and thickened mucosa. Scheible and Leopold.[5]

Radioisotope scanning image though not sharp can locate altered metabolism, metastatic tumors, etc. This modality is still in its early stages in India and since very few centers have this facility, the majority of the dental surgeons have to, make do, without using this.

Newer techniques like Thermography have been suggested by Wortzmang et al[6] to be extremely useful, especially in infections, osteomyelitis and other inflammatory conditions where the Radiography depicts minimal changes. Sinoscopy has been done by Illum and Jeppesen[7] using a Hopkins Optic system to examine the interior of the maxillary sinus and found that this investigation was extremely useful for early detection of the sinus cancer.

TAXONOMY OF SINUS PATHOLOGY

Pathologic processes affecting the sinus could be portrayed for easier understanding in the following manner:

Infection group, Cyst group, Tumors group, Trauma group, Foreign body group, Miscellaneous diseases group.

Group 1. (a) Acute Sinusitis (b) Chronic sinusitis (c) Osteomyelitis

Group 2. (a) Extrinsic cysts (b) Intrinsic cysts (c) Antroliths

Group 3. (a) Benign tumors (b) Malignant tumors

Group 4. Trauma; Le Fort I, Le Fort II, Le Fort III, etc.

Group 5. Foreign bodies and Local osseous defects

Group 6. Miscellaneous and Rare conditions.

MAXILLARY SINUSITIS OF ORAL ORIGIN

Less than 10% of the infections of maxillary sinusitis occur from tooth or displacement of tooth root into the antrum.[8] The spread of infection from periapical abscesses of maxillary posterior teeth, or the pericoronal infection from maxillary wisdom molars are the two main causes of suppurative maxillary sinusitis. The dental surgeon will encounter grossly decayed, tender on vertical percussion tooth together with pain in the malar region of the face, and foul discharge from the affected side nostril. Fever and headache will accompany. Primary cause, i.e. the infected tooth should be extracted; if oro-antral communication develops a palatal rotation flap will help to cover it (see Fig. 5.2).

Antibiotic cover using Amoxicillin, 500 mg 1 tds for 7 to 10 days or Cotrimoxazole are the antibiotics of choice. Nasal drops of Xylometazoline hydrocloride 0.1% are the drugs of choice. Menthol or Benzoin inhalation (BP) or Menthol and Eucalyptus inhalation BP are useful, aspirin 300 mg or, Asabuf® (buffered aspirin) may be given to control pain

FIGURE 5.2: Showing the method of palpation extraorally picture A, intraorally picture B and in the canine region to check for any tenderness, and thinning of bone prior to performing any biopsy through this area (Prasanna Kumar 2004, Yenepoya Dental College and Hospital, Mangalore)

and Escold to control congestion in such cases. In case of inadequate treatment chronic sinusitis may result.

Chronic Sinusitis[10]

The dental patient who suffers from chronic sinusitis presents with the pain in the maxillary teeth often with no local pathology which is evident, and the patient presents with sniffling, low grade fever and pain in the facial region in the upper part of the face (see Fig. 5.3). Radiographs and transillumination will confirm the findings of chronic maxillary sinusitis. Scully[8] mentions three ways of managing the chronic maxillary sinusitis—

- Antral Washout
- Intra-nasal antrostomy
- Caldwell-Luc operation otherwise known as radical antrostomy.

Oro-Antral Fistula

An oroantral fistula is most often caused by the extraction of maxillary teeth especially the molars or premolars which are closely related to the antrum. However, facial trauma, Neoplasms, Osteomylitis of the maxilla, and surgery to the maxilla may also occasionally lead to fistula formation. Major fistula tend to occur in the maxillary tuberosity, is caused by fracture during a difficult extraction. These are best managed by rotation flaps and details are available in any standard oral surgery books. Haanaes HR[9] analyzed 150 chronic oro-antral fistulas and found that

105.....resulted from simple extractions (70%)
29.....removal of impactions (19.3%)
11.....Cystectomies (7.33%)
7.....Endodontic surgeries (4.66%)
1.....Foreign body removal (0.66%)

Abrahams JJ and Glassberg RM[21] have linked chronic periodontal disease with two fold increase in the chronic sinusitis in their 84 patient study using the Maxillary Dentascan® GE Medical Systems. They comment that this fact should be taken into account when the implants or other surgical treatments are being considered.

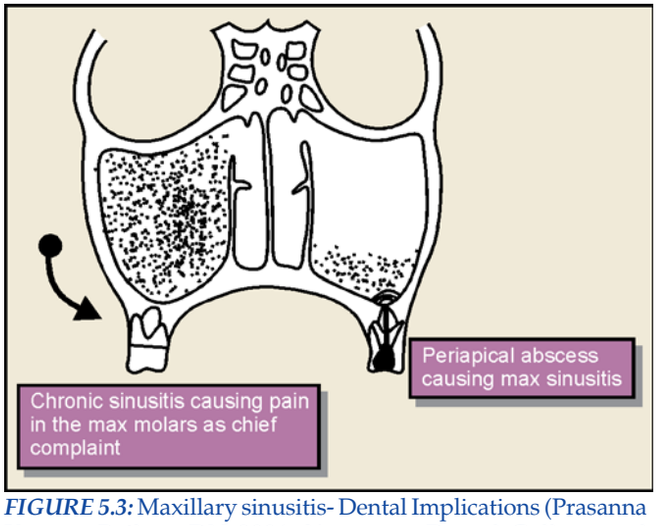

FIGURE 5.3: Maxillary sinusitis- Dental Implications (Prasanna Kumar, Bailoor DN 2004, Yenepoya Dental College and Hospital, Mangalore)

CYSTS OF THE ANTRUM

Mucosal Cysts or Intrinsic Cysts

Mucosal cysts or intrinsic cysts are of three types: (1) Mucous retention cyst or Mucocele (2) Secreting cyst

(3) Serous cyst. Of the three Mucocele may cause the erosion of antral walls.

Busaba NY and Salman SD[22] examined thirteen consecutive patients who presented with maxillary sinus mucoceles. Mechanical obstruction or allergy or both do not seem to play an important role in their formation. Endoscopic sinus surgery is a reliable therapeutic measure with a favorable long-term outcome (see Fig. 5.4).

The Extrinsic cysts in this region are Non-odontogenic cysts-Median palatine cysts, Globulomaxillary cysts. Odontogenic are—Radicular cyst, Dentigerous cyst, represent the 85.22% of the cysts occurring in the maxilla as stated by Killey HC and Kay LW.[11] These are discussed in detail in chapter on cysts (chapter no 15) Depending upon the expansion caused by the cysts surgical intervention is advocated (see Fig. 5.5).

FIGURE 5.4: PNS view of a 28-year-old female patient who was asymptomatic in the sinus region had nasal complaints. Radiograph showed cystic lesion in the right side. Diagnosis Maxillary polyp requires no Rx (Prasanna K, Bailoor DN 2004, Yenepoya Dental College and Hospital, Mangalore)

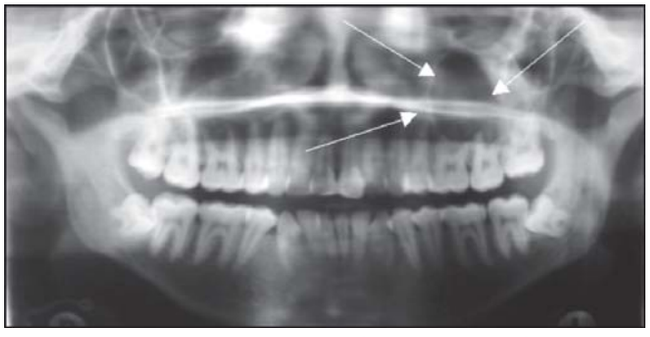

FIGURE 5.5: Showing the OPG of a 35-year-old female with slight discomfort in the left malar region. Left maxillary first molar had mesio-proximal caries and tenderness. A large dental radicular cyst was evident which had eroded into the maxillary sinus (Courtesy: Varghese Mani, GDC, Calicut 2004)

Fractures of the Maxilla

They are best managed in a hospital setting with Otolarygologist and Neurosurgeon in close proximity, with the Dental Surgeon, and good nursing care postoperative is a must for successful management.

Antroliths

Calcification of a nidus, such as blood clot, inspissated pus or mucus or exogenous foreign bodies such as cherry pits etc. If antroliths are small and discovered in routine radiographic examination not causing any problems they may be left alone and patient kept on monthly observation, if they are causing pain or any related symptoms, a standard Caldwel Luc approach seems to be enough to facilitate the removal.

MALIGNANCY AFFECTING THE SINUS

Neoplasms and other tumors related to the odontogenic apparatus.

Benign (see Fig. 5.6)

*Ameloblastoma *Calcifying epithelial odontogenic tumor (Pindborg's tumor) *Ameloblastic fibroma *Adenomatoid Odontogenic tumor (Adeno-ameloblastoma) *Dentinoma *Ameloblastic fibro-odontoma * Odonto-ameloblastoma *Complex odontoma * Compound odontoma *Fibroma (odontogenic fibroma) *Myxoma (Myxofibroma, fibromyxoma) *Cementoma—True cementoblastoma-

Cementifying fibroma-Periapical cemental dysplasia-Gigantiform cementoma *Melanotic Neuroectodermal tumor of Infancy

FIGURE 5.6: Showing a sharply defined radiopaque lesion within the left maxillary sinus. Patient was asymptomatic and biopsy revealed ossifying fibroma which was completely resected (Courtesy Varghese Mani, 2004 GDC, Calicut)

Malignant

*Odontogenic Carcinoma/Malignant Ameloblastoma/Primary Intraosseous carcinoma/Other carcinomas arising from odontogenic epithelium, including those arising from odontogenic cysts.

*Odontogenic Sarcoma/Ameloblastic Fibrosarcoma/Ameloblastic odontosarcoma

Here only two lesions will be dealt in detail.

1. Melanotic neuroectodermal tumor of infancy (MNTI)
2. Carcinoma of the maxillary antrum.

MELANOTIC NEUROECTODERMAL TUMOR OF INFANCY (MNTI)

MNTI was first reported by Krompecher in 1918[12] and called it Melanotic epithelial carcinoma. Since then the terminology has evolved and it is known by this term MNTI now.

Clinical Features

Seen within the first 18 months. There is rapid expansion of maxilla by a pigmented lesion, which is observed, through the overlying mucosa. A rounded cavity containing a developing tooth is seen on a radiograph and at operation a grey or black, soft or firm tumor mass is seen. Conservative Enucleation and Curettage will eradicate the disease. Genetic and familial trends are strongly observed and the couples who have child with this disorder are usually counseled not to have further children.

CARCINOMA OF THE MAXILLARY ANTRUM [CaANTRUM]

Occupational associations with increased risk include woodworking, shoe, textile workers and some involved in the petroleum industry. Use of home made snuff especially in the Bantu tribesmen of Africa,[11] the highest incidence of ethmoidal or antroethmoidal carcinoma is found in the Bantu of South Africa and this is claimed to be related to their use of homemade snuff. Shapiro[19] has demonstrated presence of 3:4 Benzpyrene in samples of this snuff (see Fig. 5.7).

*The relationship between wood dust and antral carcinoma has been seen in the wood workers and machinists working with African Mahoganies in the High Wycombe area, by Achesib and Cowdell et al [20] * Goren et al[13] have described an interesting case of Thorotrast induced carcinoma. These are quite uncommon accounting for less than 1 percent of all the human malignancies (St Pierre and Baker).[14]

These tumors normally have a poor prognosis because they remain undetected in the early stages and produce symptoms similar to the chronic maxillary sinusitis and in a country like India where people do not seek early treatment, by the time these tumors are detected they are in the stage IV and non-resectable. Eighty percent of the malignancies are Squamous cell carcinoma in this region.[15]

Following types of tumors occur rarely, these are melanoma, lymphoma, Olfactory nueroepithelioma, osteosarcoma, chondrosarcoma and plasmacytoma.

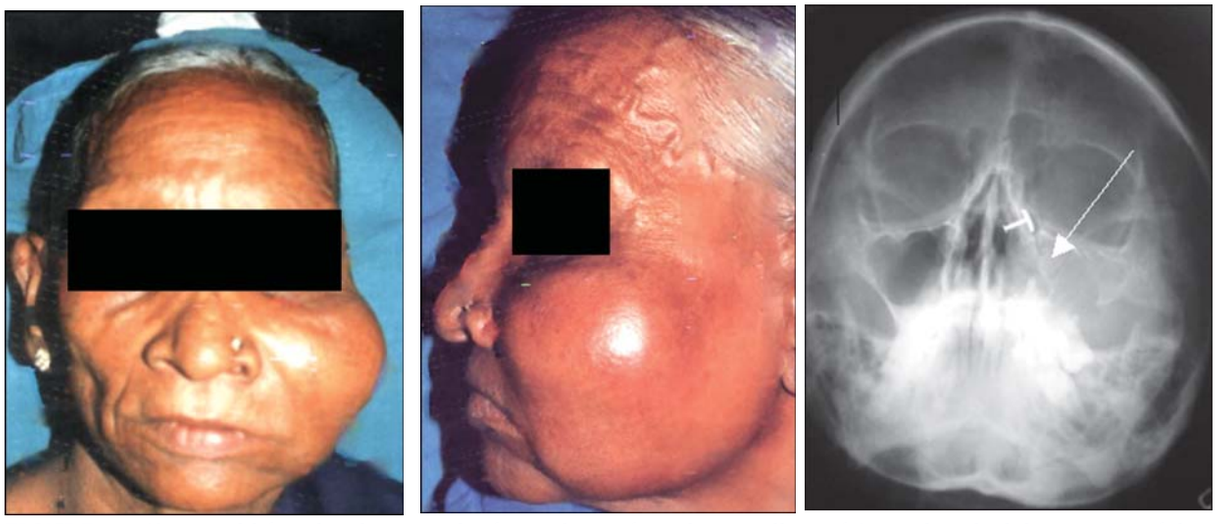

FIGURE 5.7: 60-year-old female who gave chief complaint of painless swelling of 6 months which became painful in the last ten days. PNS view gave evidence of destruction of the lateral walls of the maxillary sinus . Patient had history of snuff abuse and alcohol abuse of over twenty years duration. Caldwell Luc biopsy revealed squamous cell carcinoma invading the maxillary sinus (Prasanna Kumar, Nillofer S, Bailoor DN 2004, Yenepoya Dental College and Hospital, Mangalore)

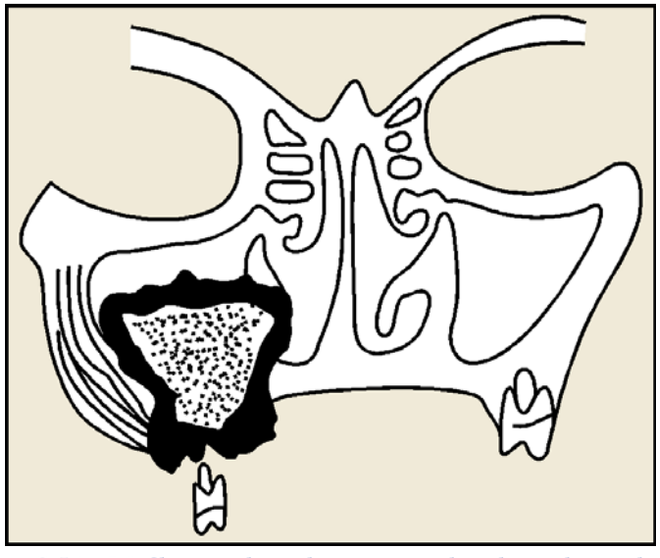

FIGURE 5.8: Showing how the tumor pushes the molar teeth out into the oral cavity. Dentist should be alert on the possibility of sinus pathology when teeth have mobility and are spontaneously exfoliated without the local pathology being suspected (Prasanna Kumar, Bailoor DN 2004, Yenepoya Dental College and Hospital, Mangalore)

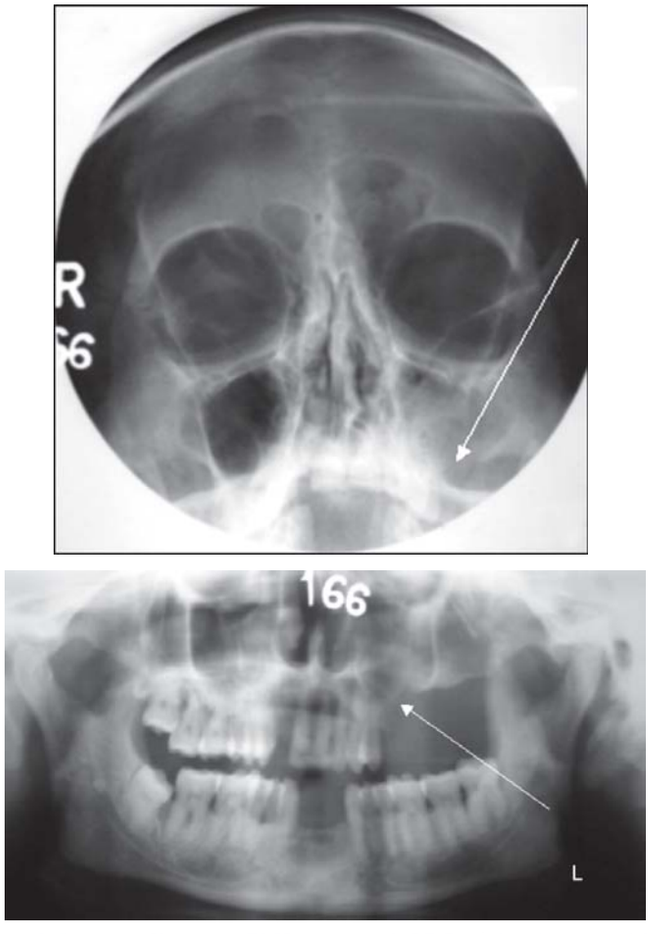

FIGURE 5.9: Showing PNS view and OPG of the 61-year-old male showing the destruction of the lateral wall and the floor of the sinus. Note how the two views complement each other in the diagnosis of sinus pathology. In OPG the innominate line destruction indicates the posterior border destruction by the malignancy . Histopathology was reported as Clear Cell Carcinoma of the antrum (Courtesy: Varghese Mani, GDC Calicut 2004)

Features

Jackson and Co workers[16] studied 77 cases and found that/ Age: range 19 to 94 years mean age=61.5 years/Male: female 4:3/Five year survival was 32%.

The tumor usually fills the sinus cavity before the specific symptoms occur. Depending on which wall of the sinus gets involved first the clinical symptoms will appear. Oral signs and symptoms including invasion of the oral cavity occur in at least 25 to 33% of the patients with maxillary sinus carcinoma (Chaudhary et al).[17] Swelling of the malar area of the face, intraoral swelling of the alveolar areas in maxillary posterior region and loosening of teeth in this area, spontaneous exfoliation and unexplained pain all point strongly towards the malignancy in the antrum (see Fig. 5.9).

The frequency of the symptoms seen is nasal obstruction, local pain, epistaxis, swelling, nasal discharge, epiphora, exophytic palatal lesion, diplopia, decreased cheek sensitivity, decreased vision in one eye, neck mass, proptosis and trismus.

Radiographically in our department we advocate two primary radiographs

1. PNS view, and
2. OPG view

   These two views complement each other.

Radiographic findings;

a. Clouding of the sinus
b. Destruction or effacement of any of the walls of the sinus. Ludman[18] is of opinion that any older patient who develops chronic maxillary sinusitis for the first time without obvious cause should be viewed with great suspicion for malignancy (see Fig. 5.9).

In Indian cities it is possible to get tomograms and CT scans for the evaluation of the antra but in smaller villages the dental surgeon will have to be happy with PNS view and Caldwell-Luc biopsy.

CONCLUSION

In Conclusion the contiguity of the maxillary sinus makes it an important consideration in the puzzling pain syndromes of the upper facial area. The importance of the role of dental surgeon in the diagnosis of simple maxillary sinusitis to the complex and elusive antral carcinoma can hardly be overemphasized. A practicing dental surgeon must be in a position to advise and interpret both PNS views and OPG for these dental patients and utilize this knowledge for effective treatment planning.

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6 Medical Emergencies in Dental Practice

INTRODUCTION

A dental surgeon should have the basic knowledge of medical emergencies likely to be encountered in dental practice. He should be competent to give the essential emergency treatment to safeguard the life of the patient until the services of a physician can be obtained. Pre-treatment evaluation and careful history of the patient forewarns the dentist of many of the emergencies in dental practice, which he may come across, and help in taking adequate precautions. Alteration in vital signs such as pulse, respiration, blood pressure etc. becomes apparent. A rapid conclusion should be drawn about the patient's general condition and severity of emergency and accordingly a quick decision should be made to bring or to maintain the vital signs at a normal level.

LA COMPLICATIONS

Malamed SF[1] mentions that most commonly used injection LA or Local Anesthetics are the safest and most effective drugs for pain control. Localized complications may arise at the site of needle penetration while systemic complications involve the entire system. Localized complications include needle breakage, paresthesia, trismus, haematoma and facial nerve paralysis, while systemic complications are psychogenic to the act of receiving an injection, allergy and drug overdose (toxic reaction).

The other conditions discussed in this chapter will be syncope, hypersensitivity reaction to various drugs, hypotension, hypertension, bronchial asthma, respiratory embarrassment and uncontrolled bleeding, apart from cardiac arrhythmias, ischemic heart disease, congestive cardiac failure, angina pectoris, epilepsy, and hypoglycemic shock.

Keur I et al[2] determined in their study that the most common medical complications were syncope and hyperventilation. Other problems were statistically not significant. But a good dentist must keep in mind that increasing numbers of elderly patients and medically compromised patients will need special preventive and corrective care if the complications are to be avoided.

SYNCOPE

Syncope is temporary loss of consciousness and posture. "Fainting" or "passing out" are the terms also commonly employed in general parlance. It is usually related to temporary insufficient blood flow to the brain. It is a common problem in the dental clinic.

Causes may be; emotional stress, pain, pooling of blood in the legs due to sudden changes in body position, overheating, dehydration, heavy sweating or exhaustion.[3]

Figure 6.1 shows how diverse the causes of syncope could be ranging from Neurologically mediated syncope

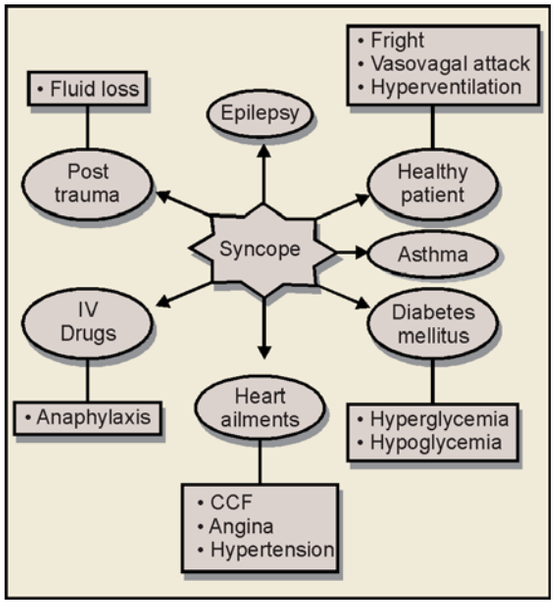

FIGURE 6.1: Figure shows the causes of syncope and how the dentist must rule out each in process of logical thinking (Courtesy: Prasanna K, Bailoor DN 2004, Yenepoya Dental College, Mangalore)

(termed earlier as vasovagal attack) to various medical problems. Usually a rapid blood pressure evaluation and pulse rate will give the dentist a good hint to the seriousness of the problem. Open communication with medical doctor and the casualty center of nearby hospital should be kept in mind.

CVS EMERGENCIES

Majority of CVS emergencies result from a combination of pre-existing pathologies and increased work requirement of heart, due to physical exertion, emotional stress or pain. Cardiac arrhythmia and alternation in BP give rise to pain, shortness of breath, headache, dizziness or syncope.[4,5]

When syncope occurs, patient should be placed in semi-reclined position with legs and thorax slightly elevated (Trendelenburg), if required, oxygen in 100% concentration should be administered with the help of facemask and BP should be monitored continuously. If hypotension persists, 5% dextrose in water or Ringer's lactate solution IV should be started. IV Mephenteramine 10-20 mg or phenylephire® 0.5-1 mg raises the BP.

Angina pectoris is a rare emergency in which the patient gets crushing pain in sub-sternal region on left side lasting for few minutes radiating to left shoulder, arm and sometimes to jaws. Tab sorbitarate 10 mg (Glyceryl trinitrate) should be given sublingually or patient is asked to inhale from broken capsule of amylnitrate (0.3 mg/dose). Nitroglycerin transdermal delivery systems [NTDS] can also be used. Nitroglycerin acts by dilation of coronary and peripheral arteries thereby decreasing cardiac work and improving blood supply to myocardium. The most probable side effects of nitroglycerin are possible drop in BP and headache.

In myocardial ischemia, severe continuous, crushing, substernal pain (may/may not be radiating to left and jaws) is present along with syncope, vomiting and extreme tiredness. Oxygen should be administered and patient is sedated with Morphine 4-10 mg IM or Meperedine 50-100 mg IM, is administered only if there is no hypotension. Morphine dilates coronary arteries and peripheral vessels decreasing cardiac work and improving myocardial oxygenation, it is also anxiolytic. Morphine is associated with hypoventilation, hence close monitoring and support of the patient is necessary. Administration of N_2O along with oxygen can also be considered as an analgesic or anti-anxiety medication.

RESPIRATORY EMERGENCIES

Bronchial Asthma

Bronchial Asthma is characterized by attacks of wheezing and dyspnea due to bronchospasm, mucosal edema or mucous secretions. These attacks are triggered by a variety of factors like allergy, respiratory infections, emotional upsets or physical exertion.

Sign and Symptoms of Bronchial Asthma

- Dyspnea
- Fragmented speech
- Paradoxic pulse
- Diaphoresis (Perspiration)
- Respiratory rate > 30/min
- Pulse > 120/min (adults)

Management of Bronchial Asthma

- Dental treatment is stopped.

- Oral cavity is cleared of materials and potential obstructions.
- Salbutamol inhaler 100-200 mg and or IV aminophylline 250-500 mg (slow infusion) should be used to relieve the bronchospasm.
- In severe asthmatic attack, subcutaneous epinephrine is administered in 0.3 ml of 1/1:1000 dilution.

This dose can be repeated every 30 minutes to a maximum of 3 doses if asthmatic attack does not resolve.

- Administration of oxygen with the help of oxygen mask if patient remains in respiratory distress after bronchodilation.
- During a refractory asthmatic attack, if patient suffers a respiratory or cardiac arrest basic cardiac life-support is initiated.

Respiratory Obstruction

Respiratory embarrassment can be due to lodging of any foreign body like tooth or removable partial denture in pharynx. Patient should be instructed to keep the mouth open and lodged foreign body is removed by *Heimlich maneuver* in which forceful elevation of diaphragm causes compression of lungs and increases airway pressure and forceful cough which helps to expel lodged foreign body.

For performing the Heimlich maneuver in a conscious patient (sitting/standing) dentist stands behind the patient and wraps his arms around the patient's waist between navel and xiphoid forcing a fist with one hand. This hand is grasped with the other hand and the fist is quickly pressed into patient's abdomen with a sharp inward and upward movement.

Unconscious patient should be place in a supine position and heel of one hand is placed between the patient's umbilicus and xiphoid process. The other hand is placed against the fist and pressure forcefully into abdomen with a sharp upward and inward thrust. Abdominal thrust may be difficult to perform if the patient is pregnant or obese. The steps are repeated if patient is not responding.

MISCELLANEOUS EMERGENCIES

In calcium deficiency condition such as tetany, patient will suffer from an attack of spasm involving skeletal muscles with lockjaw, which is treated with 10% calcium gluconate IV.

Calcium Route: IV[6]

Dosage: Magnesium intoxication: 4.5-9 mEq via IV infusion at a rate not to exceed 200 mg/minute. Hypocalcemic tetany: 4.5-16 mEq via IV infusion at a rate not to exceed 200 mg/minute. Calcium channel blocker over dosage: 5-10 mL (6.8-13.6 mEq) of 10% calcium chloride or 10-20 mL (4.65-9.3 mEq) of 10% calcium gluconate IV over 5 minutes. Hyperkalemia with secondary cardiac toxicity: 2.25-14 mEq IV. Repeat after 1-2 minutes as necessary.

Epileptic Attack

When the patient gets epileptic attack during dental treatment, the treatment is stopped immediately and the airway is maintained with the help of well-padded tongue blade, which also prevents severe injury to lips and tongue. Patient can be ventilated with oxygen if necessary.[7]

Most epileptic seizures are self-limited but if status epileptics occurs IV diazepam 1 mg every 30 sec upto 10 mg or Lorazepam upto 4 mg IV is indicated. If it is difficult to start IV line, then Midazolam IM 0.07-0.08 mg/kg body weight in patients under 60 years of age and 0.02-0.05 mg/kg body weights in patients above 60 years of age is given.

DIABETES MELLITUS

In diabetic patient, two types of emergencies can arise in dental office (see Fig. 6.2).

- Diabetic coma (Hyperglycemic coma)
- Insulin shock (Hypoglycemic shock)

Diabetic Coma (Hyperglycemic shock)

Signs and Symptoms

- Hypotension
- Tachycardia
- Fruity or acetone smelling breath
- Kussmaul's respiration: Taking deep breath through nearly closed mouth. This probably signifies hyperventilation without dyspnoea therefore without reflex opening of mouth during inspiration as in diabetic ketosis, uraemia and salicylate poisoning.

There is little a dentist can do to reverse severe hyperglycemia or treat diabetic coma, as parenteral insulin and IV fluids are necessary. So patient should be transferred to medical emergency unit immediately.

Insulin Shock (Hypoglycemic Shock)

This condition is not uncommon in dental practice.

Signs and Symptoms

- Tachycardia
- Sweating
- Nervousness
- Irritability

Management of Insulin Shock

For conscious patient few lumps of sugar/glucose or a drink of it can be tried.

For unconscious patients administer dextrose IV in 50% concentration at the rate of 10 ml/min. Other drugs for unconscious patients include 1 mg glucagon IM and 0.5 mg epinephrine IM. In children glucagon dose is reduced to 0.5 mg IM.

It is difficult to distinguish early hypoglycemic coma from hyperglycemic shock hence when in doubt there is no harm in giving small dose of oral glucose/sucrose as hyperglycemic shock will not worsen but will save hypoglycemic patient.

RARE EMERGENCIES

In ***hyper-thyroidism*** (thyroid crisis) emotional disturbances or cardiac arrhythmia may develop which should be treated by sedation and application of cold packs to reduce temperature.

In ***Adrenal insufficiency*** the most common emergency is adrenal shock/hypotension. This condition usually develops in patients who are on steroid therapy; they are treated by maintaining adequate ventilation and blood pressure. Patient is given hydrocortisone succinate 100-200 mg IV or Dexamethasone 4-12 mg IV.

ANAPHYLAXIS

It is a severe autoimmune reaction that may occur in various situations. It starts as mild irritation of the upper respiratory tract, to flushing of the whole skin to respiratory embarrassment, which can lead to syncope and collapse if untreated. In some it may become fatal.

Following agents can instigate anaphylactic reaction

- Foods—cheese, lobsters, sea food, shell fish, etc
- Pollen allergy in various times in spring to sensitized individuals
- Drugs—Aspirin, Antibiotics, NSAIDs, GA agents, LA injections, Opioids etc.
- Exercise induced anaphylaxis – in some individuals

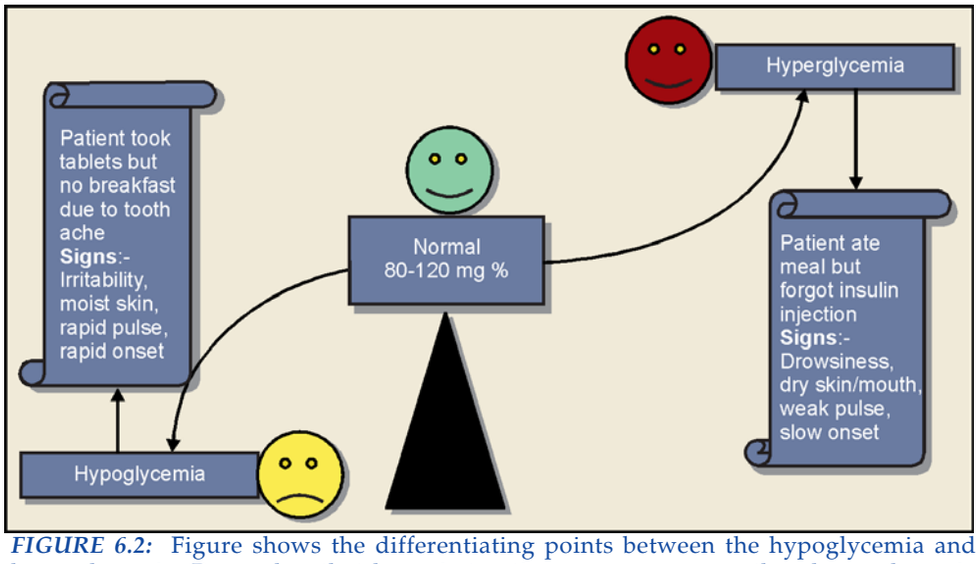

FIGURE 6.2: Figure shows the differentiating points between the hypoglycemia and hyperglycemia. Remember that hypo is ten times more common than hyperglycemia (Courtesy: Prasanna K, Bailoor DN 2004, Yenepoya Dental College, Mangalore)

Table 6.1: Common emergency conditions, their diagnostic signs and treatment

Emergency condition	*Diagnostic signs*	*Treatment*
Syncope	Pallor, sweating, cold clammy skin, giddiness, feeble pulse	Head low position, Aromatic spirit of ammonia, inhalation/2-4 ml with water. Inj Atropine 0.6 mg IV
Hypertension	Semi-consciousness/unconsciousness, headache, increased pulse rate, shortening of breath.	Tab Nifedipine 10 mg sublingually Tab Propranolol sublingually 40 mg.
Hypotension	Pallor, giddiness, sweating, lowered pulse rate (thready)	Trendelenburg position (leg up) oxygen 100% in cone by facemask. Inj Mephentremine 10-30 mg IM Inj: Ephedrine HCl 32 mg/ml/IM
Angina Pectoris	Crushing pain in left substernal region, Radiating to left shoulder, hand or jaw lasting for few minutes.	Maintain airway. 100% O_2 by facemask. Tab Nitroglycerin 0.5 mg Sublingually Nitroglycerin spray 5-15 mg Isosorbide dinitrate (sorbitarate) 5-10 mg Sublingually 5% Dextrose IV
Myocardial Infarction	Crushing pain in left substernal region with the syncope vomiting and tiredness. Patient becomes ash pale with decreasing BP, arrhythmia, thready pulse.	Maintain airway Inj Morphine sulphate 4-10 mg IM or N_2O 50% with O_2 50% Cardiovascular resuscitation Transfer to cardiac facility.
Cardiac Arrhythmia	JVP may be raised, extreme fatigue, mental confusion.	Metaprolol inj. 5 mg/ml Upto 15 mg slow IV Verapamil HCl 40 mg tabs.
Bronchial Asthma	Respiratory distress, dyspnoea, fragmented speech, paradoxic pulse, respiratory rate >30/min	Inj. 0.25 ml of epinephrine 1/1000 S/C Salbutamol 0.1 mg/dose inhalation; 2 mg tabs oral Terbutaline inhaler 250-550 Mcg inhalation Terbutaline 0.5 mg/ml subcutaneous.
Epilepsy a) Grand mal b) Petit mal	Excessive muscular activity, Loss of consciousness, muscle rigidity. Eyelids/Head moves synchronously and the seizure lasts only for few seconds, Postattack confusion.	Maintain airway and give oxygen. Protect from injury during convulsions. Sodium pentobarbital IV 60-120 mg/dose. Carbamazepine 600-1200 mg, Phenytoin sodium 300-400 valproate 750-1250 mg/ dose.
In Status Epilepticus		Diazepam 10 mg IV infusion
Hypoglycemic shock	Anxiousness, sweating, headache, diplopia, convulsions, unconsciousness, palpitations.	Dextrose 50% oral/IV
Uncontrolled bleeding	Gingival/Post extraction bleeding	Adrenaline pressure pack 1:1000 Gel foam/bone wax. Inj Vit K -IM
Anaphylactic shock	Dyspnea, wheezing, asthma, vomiting, loss of consciousness, hypotension	Inj. Dexamethasone 4-12 mg IM/slow IV Inj Adrenaline 1cc 1:1000 subcutaneous
Tetany	Spasm of skeletal muscle	Inj Calcium gluconate 10% 10cc IV drip.

- Insect stings and animal bites—bees, scorpions, wasps, spiders and snakes etc.

Signs and Symptoms

- Dyspnea
- Wheezing
- Loss of consciousness
- Bronchospasm
- Flushing
- Weak impalpable pulse
- Hypotension

Management

- Patient is laid flat and the legs are raised.
- Inj Adrenaline 1:1000 1 ml IM or
- Inj Dexamethasone 4-20 mg IM or
- Inj Hydrocortisone 100 mg IM/IV can be given depending on the severity of condition. Ambulance is called and the patient is transferred to nearest medical facility if no improvement occurs. Oxygen and IV drip can be started immediately.

HEMORRHAGIC EMERGENCIES

Hemorrhagic emergencies can be associated with conditions such as leukemia, hemophilia, thrombocytopenic purpura, anticoagulant therapy, local pathology or hypertension. First, an attempt should be made to understand the history and initial efforts such as pressure pack and bone wax should be tried and patient is shifted to hospital.

The common emergency conditions, their diagnostic signs and treatment are summarized in Table 6.1.

CONCLUSION

In the dental practice we have to treat not only the dental patients who are systematically healthy but also the dental patients suffering from various systemic diseases. Dentist should not only be familiar with various pathological conditions which can give rise to emergencies during the dental practice but also management of the emergency that may arise in the dental office.

In many cases potential drug reactions, fear and anxiety are the underlying causes for an emergency. As no drug or

Table 6.2: Drugs to be kept in dental office

Sl. No.	*Drug*	*Trade Name*	*Dose*	*Route*
1.	Adrenaline 1:1000		1 mg/ml	IM or S/C
2.	Hydrocortisone sodium succinate with water for injection	(CORT. S. HSS WYCORT)	100 mg/ml	IM or IV
3.	Glucose/Dextrose a) Powder/Gel/Tab b) Injection		50 ml of 50%	IV
4.	Pheniramine Meleate	Avil	22.75 mg/Ml 1-2 ml (for anaphylaxis)	IM or IV
5.	Glucagon 1 mg/ml	Glucagon Nova Glucagon (Torrent)	0.5-1 unit (mg)	IM or S/C
6.	Glyceryl trinitrate Tablet Spray	Angised GTN Spray Nitromint	0.3-0.5 mg 0.4 mg/dose	Sublingual Inhalation
7.	Isosorbide dinitrate	Sorbitrate, Caricap, Ditrate	5-10 mg	Sublingual
8.	Salbutamol Spray	Glaxo, salbutamol, inhalation	0.1 mg/dose	Inhalation
	Tab	Bronkotab, Asthalin Salmaplon	2 mg	Oral
9.	Terbutaline	Bricanyl	250-500 mcg	Inhalation
10.	Diazepam	Anxol, Calmpose, Paxum	10 mg/2 ml	Slow IV
11.	Oxygen cylinder with facemask			Inhalation with facemask

technique of drug administration is without risk, dentist must take all the necessary precaution to avoid emergencies and if emergency arises, he should be able to manage it till he gets the help from competent medical personnel (Table 6.1).

In India a new problem appears to be emerging. The market is flooded with spurious drugs, which are of low quality and questionable quantity standards.[8] With the result that many times unknown to the doctor the medications bought by the patient from the pharmacies may actually not work at all and worsen the patients condition. A recent report[8] states that many fake medications are being manufactured and sold all over India. Due to improvements in the printing and packaging technology it is difficult to distinguish the genuine from the spurious, even for doctors. Only the chemical analysis can actually expose the fakeness of the products. Some of these drugs are sold because they offer higher margins for the retailers and many of whom do not bother to check the veracity of the distributors.

According to the Mashelkar Committee [9] appointed by the central government the magnitude of this problem would have only increased in the last two years. They recommend that this hazard may be handled by increasing the vigilance, improving intelligence regarding the emerging spurious drugs supply and more draconian laws to punish the guilty to act as deterrents to such criminals. The spurious drug industry is becoming well established in India. According to World Health Organisation's (WHO) 2001 statistics, 35 per cent of the world's spurious drugs are produced in India, followed by Nigeria at 23 per cent. By all accounts pharmacists should stock only drugs from standard companies.

Re-usage of drugs past their expiry date is yet another menace and many such drugs are likely to find their way into the defective tendering system of the governments since only the lowest bids are accepted and middle men are least bothered about the quality.

Filling spurious drugs in used medicine bottles is another way the fakers get into the gray market. One senior administrator from Nicholas Pirmal[10] faced this peculiar problem with cough syrup. "People in north-eastern states get empty bottles from Bangladesh and refill them with a higher content of narcotics and sell them," said Mr. Sikka.

The doctors also have a stake in seeing that they report any spurious or fake drugs to the Drug Controller of the state so that this menace can be tackled at a grass roots level. Patients should also be educated that no replacement of fake drugs should be accepted if offered by the pharmacists.

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7 Bleeding in the Dental Clinic: Causes and Management

INTRODUCTION

Hemostatic mechanisms are those, which are evolved to conserve the blood loss in case of injury. Vessel wall integrity, adequate platelets, and function of the platelets, adequate levels of the clotting factors and the fibrinolytic pathway are some of the key elements of this mechanism.

Hemostatic defects are caused by abnormalities of either platelets or clotting factors. A few simple laboratory tests give important indicators to the nature of the underlying disease.

1. ***Complete blood check***— RBC, WBC, Platelet, ESR, Hb%.
2. ***Bleeding time and clotting time***
3. ***Prothrombin time***—11-15 seconds—It measures the effectiveness of the extrinsic pathway to mediate clot formation. Prolongation of more than twice the normal requires more thorough check by a specialist/ hematologist. This is also used by physicians to monitor efficacy of the oral anticoagulant therapy.
4. ***Partial thromboplastin time***—Checks the effectiveness of the intrinsic pathway to mediate fibrin clot formation. Normal PTT is 25-40 seconds. About 5 to 10 seconds above the maximum value is a result of mild bleeding abnormalities. Physicians use this test to monitor the efficacy of the Heparin therapy.
5. ***Serum for blood grouping and cross matching;*** in case transfusions are necessary.

LOCAL CAUSES

1. Infection – ANUG- Herpes simplex – recurrent herpes
2. Local Irritation – Calculus – maloccluded teeth – prosthetic appliances.
3. Rupture of blood containing bullae. The bullae appear due to systemic causes like autoimmunity but may rupture due to masticatory or bruxing stresses.
4. Congenital malformations hemangiomas, lymphangiomas, hereditary haemorrhagic telangiectasis

SYSTEMIC CAUSES

Blood Vessel Related

Infections (typhoid, malaria, typhus, bacterial endocarditis, septicemia, viral)

Chemical agents

- Increase bleeding diathesis (aspirin, indomethacin, phenylbutazone, ergotamine, other NSAIDs)
- Decrease bleeding diathesis (snake venom, EACA, Tranexamic acid)

Allergic—Anaphylactoid purpura—Henoch Scholein purpura simplex

Nutritional—Scurvy (Dentulous patients only)

Hereditary Hemorrhagic Telangiectasia (HHT)

Thrombocyte Related Disorder

- Excess (Thrombocytosis)
- Deficiency (Thrombocytopenia)
- Dysfunction (Thrombasthenia)

Thrombocytopenia

- ITP (Idiopathic thrombocytopenia)
- STP (Secondary thrombocytopenia)
- TTP (Thrombotic thrombocytopenic purpura)
- Leukemia, Multiple Myeloma
- SLE, Aplastic anemia
- Diffuse intravascular coagulation
- Hypersplenism.

Thrombasthenia

- Adherence defects von Willebrand's disease
- Bernard – Soulier syndrome
- Release defects – Storage pool disease – drug related
- Defective aggregation

Clotting Factor Related

Hereditary

- Hemophilia A (VIII),
- Hemophilia B (IX),
- von Willebrand's disease,
- Hemophilia C.

Acquired

- Vitamin K deficiencies
- Anticoagulant therapy—(Heparin-Coumarin derivatives)
- Liver diseases
- Scurvy

Miscellaneous Causes

- Aplastic anemia
- Disseminated intravascular coagulation
- Lupus erythematosus
- End-stage renal disease

Platelet Disorders

These are classified in various ways to improve understanding

- **Congenital and Acquired**
- **Quantitative and Qualitative**
- *Congenital* includes May-Hegglin anomaly, Wiskott-Aldrich syndrome, Neonatal alloimmune thrombocytopenia
- *Acquired*—Idiopathic thrombocytopenic purpura (ITP), thrombotic thrombocytopenic purpura (TTP), leukemia, aplastic anemia

Quantitative—Includes decrease in number, thrombocytopenia and increase in number, thrombocytosis.

Qualitative disorders are when number is not affected but function is impaired group is termed as thrombasthenia.

Platelets

George JN (2000)[11] have given a wonderful description of platelets. They are derived from megakaryocyte cytoplasm, which have a critical role in normal haemostasis, and in thrombotic disorders. The development of megakaryocytes is controlled by thrombopoietin, which binds to c-mpl on the surface of platelets and megakaryocytes. Thrombocytopenia and disorders of platelet function cause petechiae and mucocutaneous bleeding. Drugs may cause specific inhibition of platelet functions. According to the number of platelets in circulation, these disorders are classified into Thrombocytopenia and Thrombocytosis.

Any platelet count of below 100,000 per ml [150-300,000 per ml normal range] can be termed as thrombocytopenia. Any count above 500,000 per ml may be denoted as Thrombocytosis.

Thrombocytopenia

- *Primary*—cause is undetected. Primary group we could classify Immune Thrombocytopenic Purpura (ITP) and Thrombotic Thrombocytopenic Purpura (TTP).

  ITP involves rapid destruction of platelets due to disturbed immunologic mechanisms and TTP as a syndrome is common in young women with Thrombocytopenia, Anemia, Fever, Neurological signs and Renal failure.
- *Secondary*—cause can be established.
  - *Drug induced thrombocytopenia:* Cytotoxic drugs, Alcohol, Thiazide diuretics are the most common drugs implicated in the suppression of platelet

production by the bone marrow, Quinine, Methyldopa, Sulfonamides, heparin, gold and D-penicillamine.

- **Bone marrow depression—**Deficiency of the B-12 vitamin, Folic acid, can result in the depression of marrow function.
- **Abnormal cell infiltration** as seen in the Leukemia and in Metastatic carcinoma.
- **Hypersplenism—**Increase in the spleen size commonly associated with the portal hypertension in patients with cirrhosis leads to sequestration and destruction of the platelets. Chronic infections, Storage diseases, and neoplasms affecting the organ could be some other causes for hypersplenism.
- **Heparin** is commonly administered in the hospitalized patients for various therapies. Heparin Induced Thrombocytopenia (HIT) is a common side effect. It is a potentially catastrophic syndrome. Awareness and early diagnosis allow effective therapeutic intervention. Effective strategies include stopping heparin only and starting warfarin early. Use of recent anticoagulants is improving patient outcomes. Lepirudin, argatroban, and danaparoid are being used by the hematologists today and dentist must work closely with internal medicine and hematologists as a team and look after the orodental complications.[15]

Thrombocytosis

The increase in the number of platelets above the normal of 150,000 to 400,000 per cu. mm level may be technically termed as thrombocytosis.

This results in a hypercoagulable state in the hematological system. The platelets clump together form microthromboses and thereby deplete the clotting factors in the plasma. This leads to severe bleeding tendencies.

Fortunately rare, and in dental clinic four aspects need to be evaluated.

i. Bleeding tendencies
ii. Thromboses
iii. Bone marrow depression caused by cytotoxic agents
iv. Side effects of systemic corticosteroids

These patients are in serious condition and are hospitalized. Hematologists use systemic steroids, cytotoxic drugs or Phosphorus with P^{32} radioactive label.

Some specialists use aspirin or anticoagulants as part of therapy. Team approach is the best for their treatment.

The increase in the platelet to pathologic levels is attributed to either:

a. Reactive to some systemic problem
b. Associated with Myeloproliferative disorders

Thrombocytosis in children is common, but is usually without symptoms. Chen HL et al [3] 1999 evaluated 2910 children and 220 were found to have levels exceeding (> or = 500×10^9/L). Most of the thrombocytosis cases were due to infections, inflammatory diseases, or Kawasaki disease. No thrombotic complications were seen in any of the children.

Chuncharunee S et al (2000)[1] in a retrospective study of 126 patients with extreme thrombocytosis (defined as a platelet count > or = $1,000 \times 10^9$/L) analyzed the etiology of this condition. They determined that 55.5 percent had reactive thrombocytosis (RT); 44.5 percent had chronic myeloproliferative disorders (MPD). Underlying causes of RT were malignancy (35.7%), infection (22.9%), postsplenectomized beta-thalassemia/Hb E (15.7%), inflammation (17.1%), iron deficiency anemia (8.6%). Subtypes of our MPD cases were chronic myeloid leukemia (53.6%), essential thrombocytosis (32.1%), polycythemia vera (7.1%), agnogenic myeloid metaplasia (5.4%) and unclassified MPD (1.8%).

Aune B et al (1999)[12] have mentioned about post partum thrombocytosis, during pregnancy, changes in blood coagulation and fibrinolysis create a hypercoagulable state. The platelet count is significantly increased postpartum both after normotensive, and after pre-eclamptic pregnancies. They analyzed 22 postpartum cases and found that the peak values occur between 6-14 days, usually at a time when patients are discharged from hospital.

Atalla RK et al (2000)[2] studied 20 patients undergoing normal delivery and 25 caesarean delivery and found reactive thrombocytosis in all the patients with increase in platelet count which continued for about 24 days after the delivery. Dentists who have postponed the dental treatment of pregnant patients for medical reasons should

remember this prior to initiating surgical dentistry post-partum.

Thrombocytopathy (Thrombasthenias)

Here the number of the platelets is normal but their function is compromised.

Inherited disease is von Willebrand's disease, platelet function abnormality with Factor VIII deficiency, Bernard-Soulier syndrome and Storage Pool disease.

Acquired Abnormalities of Platelet Function

- ***Drug induced*** – Ingestion of doses like 300 mg to 1 g of aspirin results in depressed platelet function for 7-10 days. This is the normal platelet life-span. Other NSAIDs like Ibuprofen, naproxen, phenylbutazone, and indomethacin usually can impair the function for short periods between 6-10 hours. In general aspirin and other nonsteroidal anti-inflammatory drugs should be avoided if optimum hemostasis is desirable, or there is previous history of bleeding diathesis.
- ***Uremia*** – Patients with renal failure have impaired platelet function which disappears 24-48 hours after dialysis.
- ***Myeloproliferative disorders*** – Polycythemia vera, chronic granulocytic leukemia, etc.

Blood Clotting Disorders (Congenital-Acquired)

Congenital

Hemophilia A, Hemophilia B, von Willebrand's disease, Hemophilia C.

Hemophilia A or classical hemophilia is due to deficiency of Factor VIII, sex linked recessive inheritance in which females are carriers and males are sufferers. In less than 1% the deficiency is severe, between 6-30% is mild. All of the above are at significant risk.

Hemophilia B—Christmas disease is transmitted as a sex linked recessive trait. Deficiency of Factor IX is the cause, and the manifestations are similar to the Hemophilia A.

Both Hemophilia A or Hemophilia B have a variable clinical presentation. Bleeding into joints, muscles, cranium or bladder can lead to serious and often fatal complications. Dentist in most countries can readily identify the "Hemophilia Card" issued to the hemophiliacs and also some of the patients give history of regular blood transfusions.

Hemophilia C is caused by Factor XI deficiency and fresh frozen plasma usually corrects this rare type of hemophilia.

Hemophilia is diagnosed by -

a. Reduced Factor VIII C
b. APTT is increased
c. Normal Factor VIII R, von Willebrand's Factor, normal ristocetin cofactor
d. Normal PT and BT

Prior to treatment following may be administered-

- Regular replacement with Factor VIII (AHF) daily injections

  AHF can also be given one hour pre-operatively and since it is effective for only 12 hours, it is better to plan twice daily AHF injections post-operatively after major operations.
- Human freeze dried Factor VIII concentrate can be stored upto a year at 4 degrees but once reconstituted must be used immediately.
- Antifibrinolytics significantly reduce the Factor VIII requirements, *'Tranexamic acid'* is given in the dose of 1 gm orally four times daily starting 24 hours pre-operatively. *'Tranexamic acid'* can also be used topically as a mouthrinse as a 5% solution.
- Desmopressin can be given as an i.v. infusion which transiently corrects the problem by releasing the Factor VIII C and von Willebrand's into the blood.
- In locally atraumatic surgery, proper antibiotic cover and in selected cases like palate carefully designed acrylic splints should be planned before therapy.
- Hemopilia B similarly Factor IX is given at 20 units per kg body wt (Table 7.1).

Prenatal diagnosis of hemophilia: Saxena R et al (1998)[4] has stated that with the advent of recent advances in the molecular biology, it is possible to identify the multiple molecular defects. The use of polymerase chain reaction (PCR)-based linkage analysis and direct mutation detection in the chorionic villus sample obtained at 10-12 weeks of gestation has significantly improved the early diagnosis. Prevalence of the hemophilia gene in the general

population has increased recently due to reproductive fitness among hemophiliacs rendered by modern medicine practices.

von Willebrand's Disease (vWD) transmitted as an autosomal dominant mode of inheritance. This causes deficiency of Factor VIII and abnormal platelet function.

Perutelli P and Mori PG (1997)[9] explain about the human von Willebrand Factor (vWf) it is a multimeric glycoprotein present in plasma, platelets, endothelial cells and subendothelium and synthesized in endothelial cells and megakaryocytes. vWf plays a crucial role in blood clotting and platelet thrombus formation; Common presenting sign of deficiency is purpura of mucus membranes and the skin. Kadir RA et al (1998)[5] reported heavy menstrual bleeding in patients with inherited bleeding disorders, especially vWD. Quality of life during menstruation was assessed in 99 patients with inherited bleeding disorders. In dental history therefore detailed description of menstrual flow can be important indicator for bleeding problems.

Nitu-Whalley IC and Lee CA (1999)[7] did the retrospective review of 10 cases with Acquired von Willebrand's Disease (AvWD). It occurs in patients with no family history of vWD. They present with recent onset of bleeding symptoms. AvWD appears to be associated mainly with lymphoproliferative disorders, immunological conditions and neoplasia. Resolution of underlying hypothyroidism and multiple myeloma (in few cases) led to normalization of the coagulation. Treatment of the bleeding diathesis was successfully achieved with desmopressin or clotting factor concentrates.

Diagnostic lab indicators are-

a. Bleeding Time (BT) increased
b. APTT increased
c. Factor VIII R: Ag low levels of von Willebrand's factors
d. Factor VIII C and Factor VIII R: Rco (ristocetin cofactor) low levels

In presence of ristocetin the platelets fail to aggregate which results in the increased bleeding time and purpuric presentation.

Baxter Healthcare Corporation, in partnership with Genetics Institute, has introduced the first genetically engineered Factor VIII concentrate in 1992. This breakthrough offered a Factor VIII replacement that is not derived from human plasma.

ADVATE ™ rAHF-PFM, the first and only plasma/albumin-free method recombinant Factor VIII released by this corporation in 2003. Combining the Recombinate rAHF molecule with the latest advance in safety. A plasma/albumin-free method.

Acquired Disorders

Deficiency of Vitamin K dependent coagulation factors, i.e. the Factor II prothrombin, VII, IX, X are manufactured in the Hepatic cells and require Vitamin K for synthesis of their active forms.

Chuansumrit A et al (1998)[10] pooled cases from number of studies and analyzed a total number of 830 cases of vitamin K deficiency in infancy in Thailand. Of these 799 were idiopathic vitamin K deficiency in infancy (IVKDI) and 31 were secondary types. IVKDI was found in exclusively breast-fed infants most of whom (92%) did not receive vitamin K prophylaxis at birth (90%). Bleeding and pallor were the commonest features. The intra-cranial hemorrhage was 82%; the fatality rates were 24%. IVKDI is not very common in India as routinely most infants receive vaccination and vitamin K injection at birth. Some children born in remote villages may not have access to Vitamin K therapy at birth.

Liver diseases—Denninger MH (1999)[8] mentions that the liver plays a key role in the regulation of hemostasis. The liver diseases are commonly responsible for hemostatic abnormalities including decreased production of clotting factors, thrombocytopenia, platelet dysfunction, and increased circulating fibrinolytic activity. Hepatic cells play a role by producing most clotting factors and inhibitors, as well as a number of the proteins involved in fibrinolysis, and by clearing from the bloodstream activated enzymes involved in clotting or fibrinolysis, the liver protects against both bleeding and undue activation of coagulation.

The increase in portal hypertension causes sequestera-tion of the thrombocytes into the spleen resulting in relative thrombocytopenia. The hepatocellular damage results in improper production of the clotting factors, biliary disease

results in Vitamin K deficiency which in turn results in bleeding diathesis.

Table 7.1: Pharmacotherapy for various bleeding disorders

Treatment suggested	*Dosage*
Vitamin K supplementation	10 mg subcutaneously; repeat parenterally in 24 hr/do not give IV or IM
Fresh frozen plasma infusion	10-15 mL/kg, q12h; assess coagulation values after each infusion
Plasma exchange	If repeated infusions of fresh frozen plasma are required and there is evidence of volume overload
Cryoprecipitate infusion	If fibrinogen value < 75 mg/dL; use
Platelet transfusion	dosage of 2 bags per 10 kg, q12h

- Oral anticoagulants are given for protection in the thromboembolic processes the doses of which should not be altered by the dental surgeon unilaterally. Consult the physician and if dose alteration is necessary than consider hospitalization.
- Malabsorbtion syndromes results in Vit K deficiency.
- Chronic antibiotic therapy which results in destruction of the intestinal flora and consequently precipitates the Vit K deficiency.
- Prescription drugs and GI tract bleeding: Pulaníc R et al (1998)[13] caution against the prescription of ulcerogenic drugs in patients who have peptic ulcer and GERD. The most frequently taken drugs were aspirin (44.3%), piroxicam (12.3%) and ibuprofen (7.4%). In their study bleeding lesions were 1.4 times more frequently found in male users than in female users. They concluded that NSAIDs are a common cause of damage to gastroduodenal mucosa. The dental surgeon must see that the risk of drug therapy should be balanced against the risk of the disease.

Hawkey CJ (2000)[14] confirmed the findings above and in his study of 132 patients found that NSAID non-steroidal anti-inflammatory drug users had double the risk of bleeding peptic ulcers provided they were infected with *H. pylori.*

INDICATORS FOR THE DENTAL MANAGEMENT

A. All dental patients should be routinely screened for possible bleeding disorders. The medical questionnaire should include inquiries regarding easy bruisability, bleeding, or clotting problems. Excessive menstrual bleeding, frequent nosebleeds, or unusual bleeding following trauma or surgery provide clues for possible bleeding disorders. Specific questions regarding excessive bleeding following tooth extraction or periodontal surgery should be asked. Family history of bleeding disorders should be noted (Fig. 7.2).

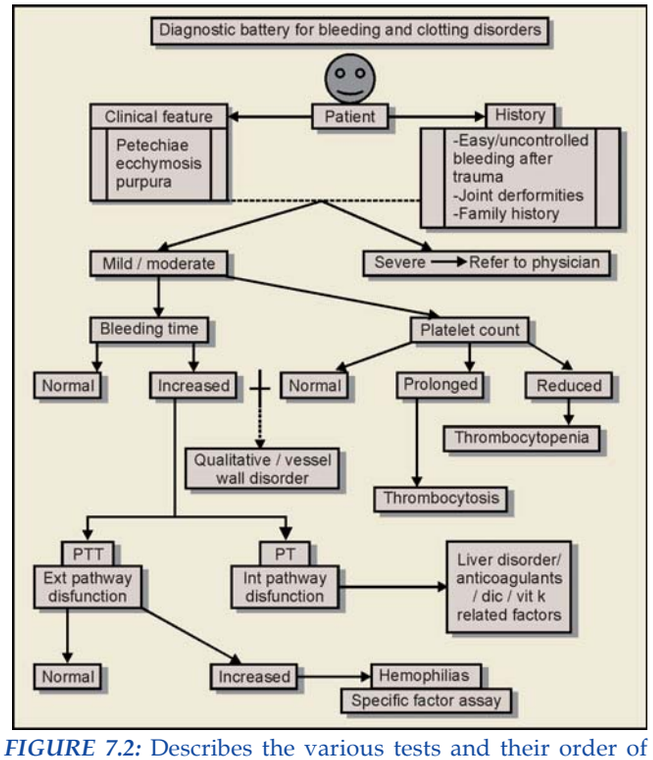

FIGURE 7.2: Describes the various tests and their order of indication for evaluation/diagnosis of various bleeding and clotting disorders (Nillofer Shabnam, Prasanna Kumar, Bailoor DN 2004)

B. Patient medications should be reviewed. Specific questions regarding aspirin ingestion and anticoagulant therapy should be asked. Patients often fail to report aspirin as a medication. Furthermore, since there are more than 200 aspirin-containing compounds commercially available, the patient may not be aware that he is taking aspirin. Revisions should be made in the dental questionnaire to allow for entry of all. Over-the-counter drugs the patient uses in order to detect aspirin containing medications.

C. During the dental examination, the dentist should be alert to the physical findings suggestive of a bleeding

disorder. Ecchymoses and petechiae are grounds for further screening.

D. Finally, prior to advanced dental surgery routine screening for possible bleeding diathesis is recommended. Test should include complete blood count, platelet count, prothrombin time (PT), Bleeding time (BT), Activated Partial Thromboplastin Time (APTT), clotting time (CT), fibrinogen level (FL), and level of fibrin degradation products (FDP).

Based on the history, clinical examination and lab tests, patients can be grouped into

- Cat-1 (low risk)
- Cat-2 (moderate risk), and
- Cat-3 (severe risk).

Low Risk Dental Patients—CAT-1

1. Patients with no history of bleeding disorders, normal examination, and acceptable lab parameters.
2. Patients with occasional bouts of bleeding, which may be attributed to local causes and lab parameters, are normal.

Moderate Risk Dental Patients—CAT-2

1. Patients on long-term anti-coagulant therapy. PT is in one and half to twice the control value.
2. Patients on long-term aspirin therapy.
   McMahon C et al (2000)[6] state that the majority of patients receiving plasma-derived clotting factors in the past specially the period between 1970s and the mid-1980s have become hepatitis C positive. Liver biopsy remains the only definitive way of staging fibrosis. They have suggested that liver biopsy is a safe procedure when done by an experienced clinician.

Severe Risk Dental Patients—CAT-3

1. Patients with known bleeding disorders thrombocytopenia, thrombocytopathy and clotting factor defects.
2. Patients with severe liver disease, past history of Disseminated intravascular coagulation (DIC), severe malabsorption syndromes, Hemophilia-A, Christmas disease etc.

DENTAL MANAGEMENT OF PATIENTS WITH BLEEDING DISORDERS

When the dentist is dealing with patient having bleeding disorder he should keep in close touch with patient's physician. None of the prescription given by him should be altered by the dentist. The dentist must select either his dental clinic or a hospital for choice of the treatment depending on Cat-1 and Cat-2 which may be treated in clinic and Cat-3 must always be dealt with in hospitals.

The nature of defect will also influence the therapeutic decision e.g. Thrombocytopenia secondary to a course of chemotherapy will gradually become better in weeks and it is worth postponing the dental treatment to get better postoperative results. On the other hand thrombocytopenia with hypersplenism is established and may not improve with time. It is more appropriate to manage such patients with platelet transfusion prior to dental therapy.

The rational use of local pressure, Ice pack, and clotting aids (gel foam, local anesthetics with adrenalin, and topical thrombin) will help routinely in managing the usual bleeding problems.

Specific Guidelines

1. Anticoagulant therapy—The dentist is balancing the risk of bleeding complication with those of underlying thromboembolic diseases. Abrupt stoppage of prescription drugs or unilateral decrease in dosage by dentist alone is not to be done. Consult patient's physician and over a period of week to 15 days work towards the goal of bringing PT to one and half to twice the control value. In OPD if the PT determination on the morning of extraction or minor surgery is found to be within the above mentioned levels treatment can be done and physician can be requested to continue anticoagulants that evening or the following morning.

   In case of unstable anticoagulant status or complex Maxillo-facial surgery this should be attempted in a teaching hospital with a consulting hematologist.
2. Long-term aspirin therapy— Aspirin ingestion increases BT minimally in most patients. However, in some patients marked sensitivity to aspirin results in BT prolongation more than 2 minutes of the normal range. In such cases third molar extraction, mandibular

nerve block or periodontal surgery must be deferred. The physician usually replaces this aspirin with some other drug and once dentist records BT within 2 minutes of the normal he may proceed.

In rare cases in spite of changing the drug or its reduction BT remains prolonged upto 10 days. This may hint a inherited defect in thrombocytic function. Hematologist's consultation is mandatory here.

3. Platelet related problems—Thrombocytopenia is diagnosed when count falls below 100 X 10^9/ltr. and oral mucosa may show petechiae, ecchymoses and post-operative bleeding.

- Drugs that effect platelet function—These drugs fall into four main groups:

I. GA- Agents, e.g. halothane.
II. Antibiotics, e.g. ampicillin, methicillin, penicillin G and some cephalosporins.
III. Anti-inflammatory drugs, e.g. aspirin, mefenamic acid, diflunisal etc.
IV. Miscellaneous, e.g. vincristine, tricyclic antidepressants, chlorpronazine etc.

- HIV infection also manifests in the dental clinic with only thrombocytopenia as a manifestation, occasionally.

- Glanzmann's syndrome (platelet aggregation problems due to defective membrane protein), Bernard — Soulier's syndrome (inherited disorder of glycoprotein which is receptor for von Willebrand's factor) and Hermansky-Pudlak syndrome (Lack of storage capacity for serotonin and adenine nucleotides which result in aggregation failure together with albinism) are all rare syndromes which may occasionally challenge the practitioners.

1. In the dental situation the drugs mentioned above should be meticulously avoided.
2. Regional block injections should be avoided.
3. For minor surgeries the level of 50,000 per cu mm and above is usually acceptable, and for major surgeries 75,000 per cu mm should suffice in emergency situation. Always have general surgeon and good nursing staff in your team in all the situations requiring expert care. Platelet rich plasma (PRP) or platelet rich concentrate (PRC) both is administered immediately before the surgery for best results. In case of immune mediated destruction of platelets the platelet transfusions do not make a significant difference.
4. Use of surgical, (cellulose based intra socket dressing), avitene, (microcrystalline collagen) may be put in socket to assist in local hemostasis.

SUMMARY

The practising dentist may encounter bleeding in the dental clinic almost everyday. The management of this condition can vary from simple pressure pack, ice pack, thrombin gel, meticulous suturing after multiple extractions, Vitamin K subcutaneous injections to complex cross matched transfusions and dedicated nursing care in a hospital setup. Whenever the dentist suspects bleeding diathesis, proper documentation of the laboratory results and written consultation with physician / hematologist is indicated.

Always prefer to use the team approach with hematologist and well-trained nursing staff for the total and correct patient management.

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8 Calcium Metabolism*

INTRODUCTION

The human body contains more calcium than any of the other essential minerals—as much as 1100-1200 gm in a 70 kg adult. That is about 1.5 percent of body weight and about 27.5 mol. The body of the infant at birth contains about 27.5 gm of calcium and calcium continuously gets deposited in the bone during the growth of the body. About 99 percent of total calcium is present in bones and teeth and remaining 1 percent in the soft tissues including blood.

SOURCE

The important dietary sources of calcium are milk and milk products, sesame seeds and green leafy vegetables. Milk is the best natural source and skim milk powder is very rich source (1.37%) of calcium. Ragi is the cheapest natural source of calcium, containing about 0.3 to 0.36 percent.

DAILY REQUIREMENTS

The FAO/WHO expert group on calcium requirements (1962) considered the question of calcium requirement and suggested practical allowances for calcium for different age groups.

Recommended allowances for calcium

Subject	*Calcium mg/day*
Infants (0-12 months)	500-600
Children (1-9 years)	400-500
Children (10-15 years)	600-700
Adolescents (16-19 years)	500-600
Adults	400-500
Pregnancy and Lactation	1000-1200

CALCIUM ABSORPTION AND FACTORS EFFECTING CALCIUM ABSORPTION

Calcium is absorbed in the intestine by two different mechanisms.

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* This chapter is not a replacement for professional dental training. Kindly verify the latest prescribing practices with your teachers and consultants prior to making real life decisions. Most values are indicative and have been checked against latest reliable sources, but the publishers and editors do not have any direct or indirect liability to the use or misuse of this prescribing information.
Prior to prescribing any medication please check that they are from ethical drug manufacturers following sound quality control practices. Follow the manufacturers directions in most prescriptions and in case of new drugs please confirm side effects, safety in children and pregnancy with the nearby-approved University hospital specialists and legitimate internet sources.

1. ***Active transport***, where calcium absorption occurs against a concentration gradient and is dependent on 1-25 dihydroxy cholecalciferol (vit D), Active transport occurs in the duodenum.
2. ***Passive diffusion***, occurs lower down in the small intestine and the amount absorbed by this process is small.

The various factors affecting the absorption of calcium are:

Vitamin D: It is essential for the absorption of calcium and the deficiency of Vitamin D impairs calcium absorption.

Phosphates: Excess of phosphates lowers calcium absorption.

Phytic acid: Phytic acid is seen in green leafy vegetables. It forms insoluble calcium salts and interferes with calcium absorption. In the past the ability of phytic acid to reduce the calcium absorption in man has been overemphasized. Many green vegetables also contain the enzyme phytase, which splits phytic acid and hence nullifies the effect of phytic acid.

pH: Calcium is well absorbed at the normal pH of the intestines, if the intestinal pH becomes alkaline, calcium absorption is lowered due to the formation of insoluble tricalcium phosphate.

Fats and fatty acids: Faulty absorption of fats leading to the presence of large amounts of fatty acids in the stools interferes with calcium absorption, as insoluble calcium salts of fatty acids are formed and excreted in the faeces.

Protein: Higher levels of proteins in the diet help to increase the absorption of calcium.

Fiber: Presence of excess of fiber in the diet interferes with the absorption of calcium.

Oxalic acid: Oxalic acid present in certain foods that forms insoluble calcium salts which is excreted in the faeces, thus lowering the calcium absorption.

DISTRIBUTION OF CALCIUM IN THE BODY

Ninety-nine percent of calcium is in the bones and teeth. Most skeletal calcium is deposited as a form of hydroxyapatite, Ca 10 (Po) 46 (OH) 2, but some amount of calcium is present as monocrystalline calcium phosphates and carbonates. Calcium concentrations of the extracellular (ECF) and intracellular fluid (ICF) are different, the ECF having a higher concentration than ICF. In the cell the cytosol concentration of calcium is very poor but the mitochondrial concentration is higher. The plasma concentration of calcium is 8.5-10.5 mg/100 ml. Calcium is present in plasma in three forms:

1. Ionized calcium—60 percent i.e. 5.4-6.6 mg/100 ml
2. Protein bound calcium—35 percent-3.5-4.4 mg/100 ml
3. Complexed calcium—5 percent as citrate, phosphate and bicarbonate 0.5 to 1.0 mg.

CALCIUM AND PHOSPHATE PRODUCT IN SERUM

The plasma phosphorus content in normal infants and children (5-6 mg) is higher than that (2.6 to 4.0 mg) found in normal adults. The Ca and P product is over 50 in normal infants and children and about 30 to 40 in normal adults. The Ca and P product is very much low in children suffering from rickets (20 to 30) and in adults suffering from osteomalacia (14-24). A high product is very important for normal ossification of bone.

If it is low, ossification does not take place. Phosphate depletion in man is non-existent under normal dietary regimens. Long-term antacid use, however, will render phosphate unabsorbed. Lotz et al [2] have described such a condition characterized by weakness, anorexia, malaise and bone pain.

PHYSIOLOGICAL FUNCTIONS OF CALCIUM

1. Calcium is the chief mineral of the bone and teeth and it gives hardness to the bone and teeth.
2. Participates in many enzymatic actions including succinic dehydrogenase, digestive enzyme like trypsin.
3. Takes part in muscular contractions.
4. Essential for the clotting of blood.
5. Regulates the permeability of capillary walls.
6. Regulates the excitability of nerve fibers, nerve centers, and neuro-muscular system.

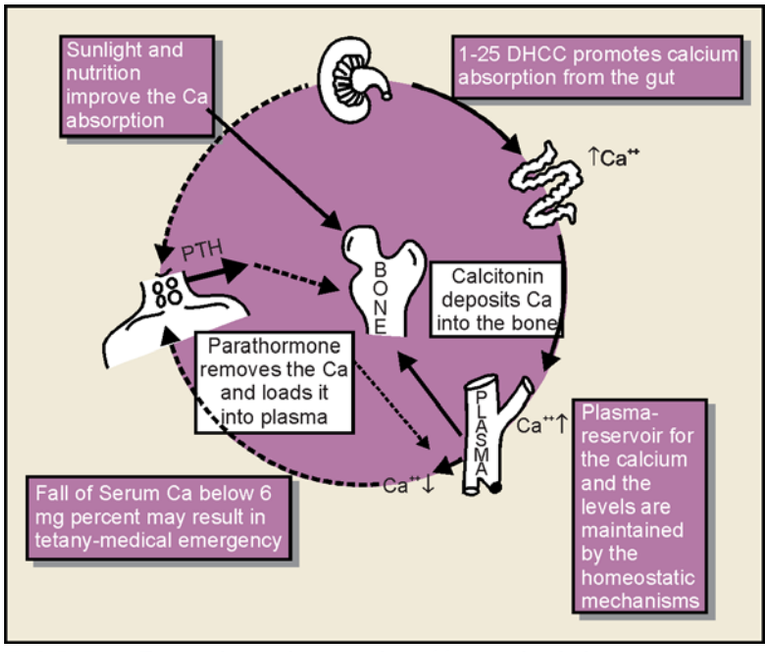

FIGURE 8.1: Figure shows the interplay of factors that help in maintaining the calcium homeostasis (Courtesy: Prasanna Kumar, Bailoor DN, 2004, Yenepoya Dental College and Hospital, Mangalore)

7. Calcium acts as the second messenger in some hormonal actions.
8. Some neurotransmitters (noradrenalin) are stored normally within the vesicles of nerve terminals. Their discharge from these vesicles requires the presence of calcium ions.

CALCIUM HOMEOSTASIS AND REGULATION OF CALCIUM METABOLISM

Ultimately the food calcium is the source of calcium in our 2body. However, the bone acts as a reservoir of calcium, supplying calcium to the serum when there is deficiency and taking out calcium from blood when it is in excess. The serum calcium, particularly the ionized calcium, is kept at a remarkable constant level and this calcium homeostasis is achieved by three hormones namely:

- Parathyroid hormone (PTH)
- Dihydroxycholecalciferol – derived from Vit D
- Calcitonin.

Calcium absorption undergoes adaptation, i.e. it is high when the calcium intake is low and decreased when the calcium intake is high. Thus, the intestine regulates the calcium metabolism by adjusting the absorption of calcium. This adjustment of absorption is made possible by changing the availability of 1.25-dihydroxy cholecalciferol – Vit D derivative.

- ***Parathyroid Hormone PTH:*** PTH is a single chain peptide with 86 amino acid residues and a molecular weight of 9500. PTH is secreted from the parathyroid gland. PTH is a ***hypercalcaemic*** hormone and it acts on bone and kidneys. PTH acts on bone and inhibits the osteoblastic activity and as a result the new bone synthesis stops and causes the increased resorption of bone. This leads to the rise of blood calcium level and to osteoporosis.
- ***Actions on the kidneys:*** PTH converts vit D into 1.25 dihydroxy cholecalciferol, calcitriol at the kidney. Calcitriol is the active form of the vit D, and acts on the intestine wall, and increases the absorption of calcium at the duodenum. Moreover calcitriol increases calcium reabsorption at the distal renal tubule by active transport.
- ***Calcitonin:*** This hormone is a 32-amino acid peptide secreted by the parafollicular "C" cells of the human thyroid. Its action is mainly on the bone. It inhibits

resorption and mobilization of calcium from bone. This leads to lowering of serum calcium content. This is a ***hypocalcaemic*** hormone.

The serum calcium level is a major factor controlling the secretion of PTH and Calcitonin. When serum calcium level is elevated it stimulates the "C" cells of thyroid gland and stimulates the increased secretion of calcitonin. Calcitonin action will bring the serum calcium level back to normal level. In contrary the fall in serum calcium level stimulates the parathyroid gland to secrete more of PTH. Once PTH level increases it brings about a rise in serum calcium level by acting at bone, kidneys and intestine. The inter-relationship between the serum calcium level and secretion of calcitonin with PTH is summed in Figure 8.1.

DISORDERS OF CALCIUM METABOLISM

Effects of Calcium Deficiency

The dietary deficiency of calcium in children will lead to:

1. Decreased rate of growth.
2. Negative calcium balance.
3. Loss of calcium from bone leading to the development of osteoporosis.
4. Hyperplasia of parathyroid gland.
5. Hyper irritability and tetany leading to death.

In adults calcium deficiency will lead to osteoporosis, it is a condition in which decalcification of the bone occurs. Fracture of bones may happen even due to minor accidents.

Hypoparathyroidism

Hypocalcaemia may result from hypofunction of parathyroid gland. This may occur due to accidental damage or removal of the parathyroid glands during surgery of the thyroid or in cancer larynx. When the parathyroids are absent the serum calcium level with the ionized fraction falls rapidly. The outstanding sign of Ca++ deficiency is tetany. When serum Ca++ level falls, the irritability of nerves as well as neuromuscular junction rises and the muscle contracts when subjected to subthreshold stimulus and even spontaneously. Basic feature of tetany is uncontrolled, painful prolonged contraction (spasm) of the voluntary muscles.

The important clinical features are:

- *Accoucher's hand*. There is muscular spasm leading to uncontrolled prolonged flexion of the metacarpophalangeal joints while fingers remain extended.
- *Laryngsmus stridulous (laryngeal stridor):* There is spasm of the larynx, the breathing stops and the patient tries violently to inspire. After sometime the spasm disappears, the air enters the larynx with a characteristic crowing sound, during inspiration, after a temporary stoppage, is called laryngeal stridor.
- *Chvostek's sign:* Taping the facial nerve at the ramus of the mandible in front of the ear, produces the painful spasm of the facial muscles.

Treatment

1. Intravenous injections of soluble calcium salts.
2. Large dose of vitamin D and increased amount of dietary calcium are long-term measures.

Hyperparathyroidism leading to Hypercalcemia

High level of serum PTH leads to excess bone resorption and brings about an increase in serum calcium level. The important signs of hypercalcemia are:

- Neural sedation as manifested by drowsiness
- Renal stones and osteoporosis leading to development of bony cysts and pathological fractures.

The main uses of calcitonin so far are in the treatment of hypercalcemia and Paget's disease.

Hypercalcemia

The causes include thiazide drug therapy, Addison's disease, immobilization, sarcoidosis, multiple myeloma, malignancy of bone, hyperparathyroidism, and vitamin D intoxication. Calcitonin acts rapidly and can reverse severe hypercalcemia, which might otherwise be fatal due to renal failure or cardiac arrhythmias (Rose and Kaye).[1]

Paget's Disease

In the early stages especially, osteoclastic activity predominates and may cause hypercalcemia and severe bone pain. These can both be corrected by giving calcitonin. Salmon calcitonin is less strongly antigenic than porcine

but antibody production is a limiting factor in the prolonged treatment necessary for Paget's disease.

Disodium etidronate: Etidronate is absorbed into hydroxyapatite crystals and slows both their rate of growth and resorption. This reduces the rate of bone turnover characteristic of Paget's disease and etidronate is probably the treatment of choice since it can be given in repeated courses without diminished activity.

Osteoporosis

The bone is one of the most dynamic tissues in the human body constantly remodeling, constantly changing in density to meet the demands of the body. An athlete who exercises regularly will have healthy and correct density bone. An executive who sits on his computer whole day without physical exercise will show lesser density of bone together with mild atrophy of muscles too. Before thirty years of age usually the building up activity of bone is more than the resorbtive activity. Older age sees the bone changing both in the matrix formation (more brittle) and less dense (likely to fracture).

Osteoporosis is not inevitable part of old age, and may be detected early and treated with reasonably good results.[3] How many Indian it affects today exactly is still not documented yet but studies are proceeding. The dentist can play a crucial role in diagnosis of osteoporosis. This occurs more commonly in postmenopausal women, more in slender women and those who have a family tendency to get it. Both the tobacco smoking and alcohol drinking tends to have negative effect on the calcium deposition. If untreated it leads the person becoming stooped and smallest of falls results in the fractures (see Fig. 8.2).

Tandon N et al[4] report that there is a definitive need for the Indian Values for bone density to be established. They also opine that childhood malnutrition may play a crucial role in decreased bone density in persons apparently consuming nutritious food and doing regular exercise in sunshine.

Pande KC[6] established a reference database of bone mineral density in the Indian women and men using digital X-ray radiogrammetry in Nagpur. To his surprise he found that about 50 percent women and 36 percent men, over 50 years of age, were noted to have low bone

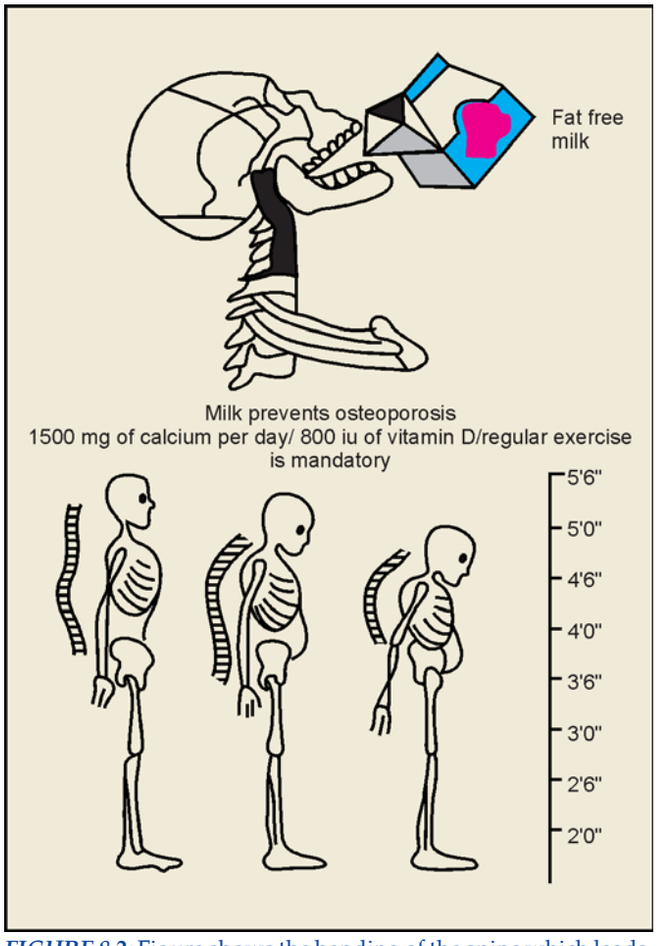

FIGURE 8.2: Figure shows the bending of the spine which leads to a stooped figure which is characteristic for osteoporosis - taken from various sources

mass. The observations of this study suggest that there is higher prevalence of low bone mass in the Indian population compared to the western population.

In South Indian women Anburajan M et al [7] have tried to establish normative data regarding bone mineral density using Dual-Energy X-ray Absorptiometry (DEXA). Many such studies will help to highlight that we need Indian standards to establish the comparative levels of osteopenia rather than depend on western textbooks for the Caucasian standards.

Diagnosis can be made on combination of -
Clinical examination, Dual-Energy X-ray Absorptiometry (DEXA), use of orthopantomograph and a detailed nutritional history. Serum biochemical values of calcium,

phosphorus and alkaline phosphatase may also help the clinician to zero in on the causative factors.

Taguchi A et al [9] evaluated the usefulness of width and morphology of the inferior cortex of the mandible on panoramic radiographs in the diagnosis of postmenopausal osteoporosis. They found that it was a viable method to supplement the diagnosis.

Law AN et al [10] evaluated dental radiographs for signs of osteoporosis using four methods: (i) fractal dimension, (ii) microdensitometry, (iii) pixel intensity and (iv) panoramic analysis of cortical thickness. They found all methods effective but pixel intensity measurement was established by them to be the best in this group for diagnosis of osteoporosis.

Most physicians rely on DEXA or Dual-Energy X-ray Absorptiometry. This test is quite reliable and takes about 10-15 minutes and measure bone density of hip and spine. It uses less radiation than an average chest radiograph.

Doggrell SA [8] has presented an excellent review on pharmacotherapy for osteoporosis. Calcium and Vitamin D together form the basic two pronged attack on osteoporosis. Combination of estrogens and raloxifen both help to prevent the bone loss in postmenopausal women. Calcitonin and bisphosphonate alendronate prevent fractures and help decrease loss of calcium.

Triparatide is one agent, which is known to increase bone formation as compared to above-mentioned agents, which prevent the resorbtion.

According to Kung AW et al[5] Raloxifene increases bone mineral density (BMD) and decreases biochemical markers of bone turnover. Considering its efficacy and safety, it is under serious consideration for routine clinical usage in Asian women. The recommended dose is 60 mg per day.

New or alternative drugs for the treatment of osteoporosis include tibolone, new SERMs, androgens, growth hormone, and insulin-like growth factor-1 and stontium ranelate.[8]

Dentists must recommend following to their patients;

- Nutritional counseling with the following — drink at least five glasses of milk, five cups of curd (Dahi), Sardines and other fish, and Broccoli amongst vegetables.
- Below 60 years of age at least additional 1000 mg of calcium per day (two tablets of calcium 500 mg per day at least) after 60 years of age three such tablets a days making it about 1500 mg per day.
- Mild to moderate exercise morning and evening, and getting at least one hour of sunshine in a day
- A practicing dentist must not prescribe hormone replacement therapy or any of the drugs like Alendronate or Raloxifene. This is best left to the specialists with whom a constant referral relationship may be maintained for better patient care.

For more information about osteoporosis, contact web site, http://www.nof.org

SUMMARY

Some of the factors controlling blood calcium can be:

1. Dietary intake of vitamin D and calcium.
2. Parathormone secretion promotes removal of calcium from the bones and raises the blood calcium level. PTH also accelerates the conversion of vitamin D into 1-25 DHCC, which increases absorption of calcium at intestinal level and decreases renal excretion of calcium.
3. Calcitonin opposes the action of parathormone and lowers the blood calcium mainly by increasing deposition of calcium in the bones.
4. Absorption of calcium may also be depressed in intestinal diseases characterized by malabsorption, i.e. steatorrhoea.
5. Excretion of calcium is enhanced in some chronic renal diseases.
6. There is a greater demand for calcium during pregnancy and lactation, hence requiring nutritional support, failing which removal of calcium may be excessive from the bone by these mechanisms.
7. Ionizable calcium is depressed when there is alkalosis. This in turn may be due to overbreathing (losing CO_2) or vomiting (losing gastric acid) and can lead to tetany.
8. Osteoporosis is evident increasingly in the menopausal females and males due to long-term negative calcium balance. Androgen and estrogen replacement therapies have been alternately used, instead of high calcium intake together with strontium and NaF ingestion.

ALKALINE PHOSPHATASE, AND LEUKOCYTE ALKALINE PHOSPHATASE

When diseases like fibrous dysplasia, primary or secondary hyperparathyroidism, osteoporosis, multiple myeloma, osteogenic sarcoma, or metastatic malignancy are suspected, it is customary to order serum calcium, phosphorus and alkaline phosphatase estimation as initial screening procedures.

Auto analyzer are becoming increasingly available in laboratories in Urban India and routine estimations should be encouraged of all variables blood chemistry profiles, and abnormal values may be discovered in the absence of signs or symptoms suggestive of bone disease. Some of the other conditions like kidney and liver disease are likely to be associated with such abnormalities

Patients with histologically diagnosed giant cell lesions of the jawbones are frequently referred for serum calcium, phosphorus, and alkaline phosphatase determinations to rule out the possibility of hyperparathyroidism. Radioassay techniques for parathormone are now available in big cities in India and allow for more specific diagnosis of increased parathyroid activity.

Alkaline Phosphatase

Occurs mainly in osteoblast and in other tissues. Increases in the serum concentration of this enzyme are seen in increased osteoblastic activity but also seen in association with obstructive liver disease and a variety of miscellaneous conditions such as malignancy or abscess of the liver, amyloid disease, leukemia, and sarcoidosis. In the absence of evidence of liver disease, the rise is usually assumed to be the result of increased osteoblastic activity.

Osteoblastic activity is seen in sclerosing bone lesions and in lytic bone lesions. Increases are also observed in periods of rapid bone growth in infancy and childhood, during pregnancy, and in healing fractures. In general alkaline phosphatase is raised in obstructive liver disease of both intra- and extrahepatic origin.

As with other enzyme assays direct measurement is not possible, and values are expressed as "units" in terms of the level of enzyme activity (e.g. by measuring changes in substrate or some product of the reaction) and not in micrograms.

The normal values for serum alkaline phosphatase are 1 to 4 Bodansky units or 3 to 13 King-Armstrong units/dl and 30 to 110 IU (international units) per 100 ml.

Alkaline phosphatase exists as several isozymes, originating in bone, biliary duct epithelium, placenta and intestine. More than 30 percent heat stable alkaline phosphatase suggests a hepatic origin for the increased enzyme, less than 30 percent suggests a bone origin.

Alkaline phosphatase is also present in the granulocytes of circulating blood, and a histochemical technique is used to demonstrate this enzyme [leukocyte alkaline phosphatase (LAP)] as a diagnostic aid in the differentiation of myelogenous leukemia from the florid leukocytosis (total WBC may be as high as 100,000 cells/c.mm) seen in some pyogenic infections, tuberculosis, drug intoxications, and malignant disease encroaching on the bone marrow. In acute and chronic leukemias LAP is usually low; the enzyme is absent from normal and malignant cells of the lymphocytic series; it is increased in mongolism.

TRADE NAMES OF SOME CALCIUM FORMULATIONS AVAILABLE IN INDIA

1. Shelcal kid-tabs ® Elemental calcium 250 mg + vit D3 125 iu chewable tabs.
2. Calcinova tabs ® Chewable calcium 500 mg.
3. Calcium Sandoz ® Chewable calcium 500 mg.
4. Sigma Calvit ® Cal glucono-lacto-bionate 140 mg +vit B12 50 mcgm_Vit D3 5000 iu per ml injection.
5. TriCal-D® syrup – calcium lacto-bionate 56 mg, Cal gluconate 60 mg, Cal lactate 88 mg, cal glycerophos 15 mg vit D3 400 iu per 5 ml syrup.In children give 5-10 ml tid. All other tablets are normally given once a day 500 mg tab in children.

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Yadav NS, Bailoor DN

9 Oral Manifestations of HIV Infection

INTRODUCTION

AIDS (Acquired Immunodeficiency Syndrome) is one of the dreaded disease of the last century which is posing a threat to the very existence of human race today. No disease has struck with such a seriousness as AIDS.

"Mouth is the mirror of health" so it is the Dental Surgeon who has a very important role to play, in the early diagnosis of HIV / AIDS by picking up the early oral clinical manifestations.[10]

HISTORICAL REVIEW

Disease syndromes similar to the clinical manifestation of AIDS have been described in the ancient Ayurvedic literature. *Sushrutha* in 800 BC and later *Charaka* and *Vagbhatta* described conditions as "Loss of muscle mass, fever, skin eruptions and ulcers, complexion changes, neurological disorders, exhaustion, coma and death, and stated that in irremediable stages treatment should be given up".[3]

Greenspan JS (1995)[4] in his historical review stated that several American missionary doctors and surgeons in 1960s who had operated in unhygienic conditions in Africa without gloves often acquiring injuries contaminated with patients blood are known to have turned sick and had died from an unrecognized syndrome similar to the presently known clinical picture of AIDS.

In mid-1981, AIDS was first recognized when unusual clusters of Pneumocystis carinii pneumonia and Kaposi's sarcoma was reported in young previously healthy homosexual men in New York city, Los Angeles and San-Francisco.[4]

Greenspan D et al (1992)[13] stated that the homosexual men from New York who visited Haiti perhaps became infected and carried the disease to USA.

Nahmias AJ et al[14] in their historical review stated that human immunodeficiency virus (HIV) infection is thought to have originated in Central Africa at the same time or even before AIDS was diagnosed in the United States. Serum samples collected from Africans at earlier periods showed the presence of antibodies reactive with HIV-1, which suggested infections were present in Africa before.

It was in May 1983 that Luc Montagnier group at the Louis Pasteur Institute, Paris had succeeded in isolating a retrovirus from West African patients with persistent generalized lymphadenopathy, which was a manifestation of AIDS. He named the virus Lymphadenopathy Associated Virus (LAV) and sent Gallo a sample in September 1983. In May 1984, the American group led by Robert Gallo confirmed the finding of French group and they named the virus as Human T-cell lymphotrophic virus III (HTLV-III).[5]

In 1986, the international committee on Taxonomy of virus ignored both LAV and HTLV-III and proposed the

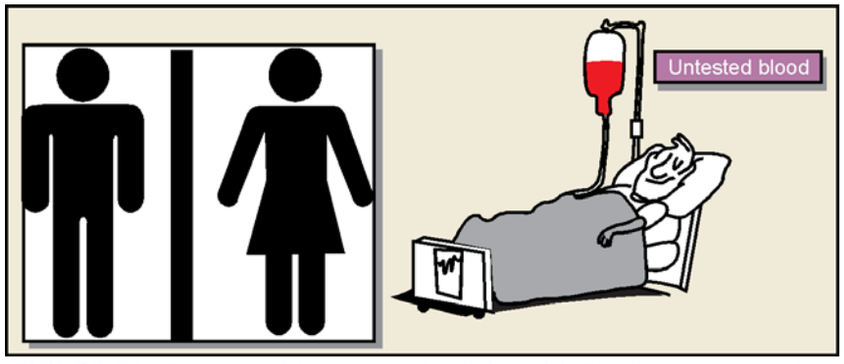

FIGURE 9.1: Unprotected heterosexual activity with Prostitutes (sex workers) and blood obtained from tainted sources results in the maximum number of patients getting exposed to HIV virus in the Indian scenario (*Coutesy:* Bailoor DN, 2004, Yenepoya Dental College and Hospital, Mangalore)

name HIV (Human Immunodeficiency Virus) and this name has become universally accepted.[5]

Deborah Greenspan et al (1992)[13] stated that similarity of HIV-2 to a virus endemic to African green monkeys, simian immunodeficiency virus (SIV) revealed speculation that human retrovirus might have evolved from its simian relative.

WHO and NACO (India) reported the first case of AIDS in India from Mumbai in 1986.[6]

TRANSMISSION OF HIV

World Health Organization (WHO) and National AIDS Control Organization (NACO)[6] enumerated the different modes of transmission of HIV.

1. Sexual intercourse (anal/vaginal/oral) with an infected partner.
2. Transmission with infected blood, blood products, organ tissue transplantation and artificial insemination (Fig. 9.1).
3. Contaminated needles and syringes
   - IV drug abusers
   - Professional blood donors working through seedy places
   - Needle prick injuries (Fig. 9.2)

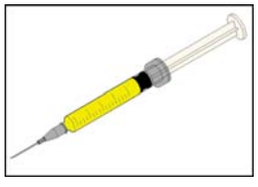

FIGURE 9.2: Unintentional needle stick injuries is one of the main mode of infection for health care professionals

4. From infected mother to her child, i.e. perinatal transmission
   - During gestation (in utero)
   - During delivery (intrapartum)
   - Postpartum through breastfeeding

DEFINITION OF AIDS

Centres for disease control (CDC) in 1993 had defined Acquired Immunodeficiency syndrome (AIDS) as the occurrence of one or more group of life-threatening opportunistic infections, malignancies, neuralgic diseases and other specific illness in patients with human immunodeficiency virus (HIV) infection and/or with CD4 counts less than 200/mm^3. Centers for disease control stated that this definition was a surveillance definition that was established to track the incidence of this disease and the relative occurrence of diseases that are likely to occur in severe immunosuppressed individuals. They quoted, that, in those parts of the world where CD4 enumeration is not readily available, Clinical diagnosis, in conjunction with serologic tests for HIV, could be used to define patients with AIDS and to track the spread of the epidemic.[5]

INFECTION AND PROGRESS OF HIV INFECTION/AIDS

The natural history of HIV infection begins as soon as virus enters the body of a susceptible host through any of the routes of transmission. Clear knowledge of natural

history of a disease help in identifying the stage and appropriate intervention to prevent or control the disease.

WHO and $NACO_2$ India (1997)[6] stated that the events after the exposure, HIV virus follow a reasonably predictable chronological order.

They proposed various stages in the natural history of HIV infection (Table 9.1).

A. Acute infection
B. Early asymptomatic infection
C. Late asymptomatic infection
D. Symptomatic infection

Table 9.1: Stages of HIV infection[5]

Stage	*Clinical manifestations*	*CD4 cell count*
Acute	Mononucleosis like illness	Normal
Early	Asymptomatic or persistent Generalized lymphadenopathy Aseptic meningitis	> 400
	Dermatologic manifestations	> 400
Middle	Asymptomatic or persistent Generalized lymphadenopathy Thrush Hairy leukoplakia Idiopathic	200 - 400
	Thrombocytopenic purpura	200 - 400
Late	Opportunistic infections Malignancy Wasting Dementia	< 200

WHO CRITERIA OF HIV INFECTION

WHO and NACO 1997 [6] suggested the criteria for HIV infection both in adults and children. This criteria was based on the clinical disease and was classified into cardinal findings, characteristic findings and associated findings.

Cardinal Findings

Adults

a. Kaposi's sarcoma
b. Pneumocystis carinii pneumonia
c. Toxoplasma encephalitis
d. Esophageal candidiasis
e. Cytomegalovirus retinitis

Children

a. Kaposi's sarcoma (rare in children)
b. Pneumocystis carinii pneumonia
c. Lymphoid interstitial pneumonitis
d. Esophageal candidiasis

Characteristics Findings

Adults

a. Oral thrush
b. Oral hairy leukoplakia
c. Miliary, extrapulmonary or non-cavity pulmonary tuberculosis
d. Cryptococcal meningitis
e. Herpes zoster, multidermatomal in less than 50 years of age

Children

a. Severe pruritis (itching without lesion)
b. Non-Hodgkin's lymphoma
c. Recurrent bacterial/viral infection
d. Herpes zoster, past or present
e. Progressive neurological disease

Associated Findings

Adults

a. Weight loss more than 10 percent
b. Fever (continuous or intermittent > 1 month)
c. Diarrhoea (continuous or intermittent > 1 month)
d. Generalized extrainguinal lymphadenopathy
e. Skin infection (severe recurrent)
f. Cough for more than 1 month
g. Dermatitis

Children

a. Neurologic findings (dementia)
b. Focal motor deficits. Neuropathy
c. Progressive headache
d. Drug reactions (previously not seen)
e. Fever (continuous/ intermittent >1 month
f. Generalized lymphadenopathy

ORAL MANIFESTATIONS

Oral and perioral lesions are common in HIV infected patients and most oral lesions appear as early signs.

Scully C et al (1991)[15] have documented oral disorders in HIV disease as which were more common and less common in HIV patients.

A. INFECTIONS

	More Common	*Less Common*
FUNGAL		
1.	Candidiasis (Fig. 9.3A)	1. Aspergillosis
2.	Histoplasmosis	
3.	Cryptococcus neoformans	
4.	Geotrichosis	
BACTERIAL		
1.	HIV gingivitis	1. Mycobacterium avium intracellulare
2.	HIV periodontitis (Fig. 9.3B)	2. Klebsiella pneumonia
3.	Necrotizing gingivitis	3. Enterobacterium cloacae
4.	Escherichia coli	
5.	Salmonella enteritidis	
6.	Sinusitis	
7.	Exacerbation of apical infection	
8.	Submandibular cellulitis	
VIRAL		
1.	HSV	1. HPV
2.	VZV	2. CMV
3.	EBV(including hairy leukoplakia)	

B. NEOPLASMS

1.	Kaposi's sarcoma	a. Non-Hodgkin's lymphoma
		b. Squamous cell carcinoma

C. NEUROLOGICAL DISTURBANCES

a. Paresthesis
b. Facial palsy
c. Hyperesthesia
d. Dysphagia
e. Trigeminal neuralgia

D. MISCELLANEOUS

a. Recurrent aphthous ulcer
b. Progressive necrotizing ulceration
c. Toxic epidermolysis
d. Delayed wound healing
e. Thrombocytopenia
f. Xerostomia and Sicca type syndrome
g. HIV embryopathy
h. Hyperpigmentation
i. Granuloma annulare
j. Exfoliative cheilitis
k. Lichenoid and other drug reactions.

Revised Classification

WHO collaborating center and EC clearing house (London) revised the classification of oral lesions associated with HIV infection on September 1992 which was reported by Williams DM (1993)[16].

Group I: Lesions strongly associated with HIV infection

Group II: Lesions commonly associated with HIV infection

Group III: Lesions uncommonly associated with HIV infection

Group I: Lesions Strongly Associated with HIV Infection

- Candidiasis—Erythematous, Pseudomembranous
- Hairy leukoplakia
- Kaposi's sarcoma
- Non-Hodgkin's lymphoma
- Periodontal diseases—Linear Gingival Erythema, Necrotizing Gingivitis/Periodontitis

Group II: Lesions Commonly Associated with HIV Infection

- Bacterial infections—Mycobacterium avium, Mycobacterium tuberculosis
- Melanotic hyperpigmentation
- Nectrotizing ulcerative stomatitis
- Salivary gland disease—Dry mouth (decreased salivary flow) or unilateral and bilateral swelling of the salivary glands
- Thrombocytopenic purpura
- Ulceration (not otherwise specified)
- Viral infections—Herpes simplex virus, Human papilloma virus, Condyloma acuminatum, Focal epithelial hyperplasia (FEH), Verruca vulgaris, varicella zoster virus (herpes zoster- varicella)

Group III: Lesions Uncommonly Associated with HIV Infection

- Bacterial infections—Actinomyces israelii, *Escherichia coli*, Klebsiella pneumonia
- Cat scratch disease
- Drug reactions
- Epithelioid (bacillary) angiomatosis
- Fungal infections other than candidiasis
- Neurological disturbances
- Recurrent aphthous ulceration (RAU)
- Viral infections—Cytomegalovirus, Molluscum contagiosum

Incidence and Prevalence of Oral Manifestations

Van der Waal et al (1991)[7] did a study on oral manifestations in 100 HIV infected individuals. In their study they observed that 56 patients had candidasis, 27 suffered from periodontal diseases, Hairy leukoplakia was diagnosed in 15 patients, Kaposi's sarcoma in 4 and 20 patients had no abnormalities.

Michael Glick (1994)[8] conducted oral examination on 454 HIV infected persons for assessment of immunosuppression and associated diseases. He co-related the CD count with different oral manifestations. From their study they reported that in candidial infected persons CD4 cell count was below 149 cells/mm^3, in oral hairy leukoplakia CD4 count was 143.3 cells/mm^3, 126 cells/mm^3 in xerostomia, 51.8 cells/mm^3 for NUP, 98.7 cells/mm^3 for HSV infection, 66.6 cells/mm^3 for Kaposi's Sarcoma and 33.7 cells/mm^3 for major apthous ulcers.

Anil SJ et al (1997)[1] conducted a study in 96 HIV infected patients in Indian population. In their study candidasis, periodontal disease, hairy leukoplakia, apthous ulcers and tuberculous ulcers, squamous cell carcinoma and non-healing extraction wound was observed. From their study they concluded that low prevalence of hairy leukoplakia and absence of Kaposi's Sarcoma had suggested variations in frequency of oral lesions among AIDS patients in Asia.

Trevor M Arendrof et al (1998)[2] conducted a study on oral manifestations of HIV infection in 600 South African patients. From their study they concluded that 60.4 percent of cases had one or more lesions. Candidial lesion was seen in 37.8 percent, gingival and periodontal lesion in 8.5 percent, Hairy leukoplakia in 19.7 percent was observed. Less commonly recorded lesions included oral ulcerations in 2.9 percent and Kaposi's sarcoma was diagnosed in 1.5 percent.

Yadav NS and Praveen BN (2000)[9] in their study of 52 HIV positive patients found that 76.92 percent had transmission by multiple sexual partners of heterosexual

A

B

FIGURES 9.3A and B: A. Severe Candidiasis of the tongue with burning and pain. B. Advanced Periodontitis in young patient. Local factors make the diagnosis complicated. These two diseases were the commonest in the Indian series amongst the HIV positive persons who sought dental consultation (Coutesy: Nillofer S, Prasanna K, Bailoor DN, 2004. Yenepoya Dental College and Hospital, Mangalore)

type. Diagnostic criteria suggested by Greenspan et al (1992) and European Community clearing house and WHO were used to diagnose the oral lesions. In this study it was observed that the primary mode of transmission is through sexual contact and predominantly seen among heterosexual who had practiced sex with multiple partners. The most common age group observed in this study is in between 30 to 39 years and next common age group is between 20 to 29 years. Males were more affected than females and about 31 of the 52 cases had a Tuberculosis infection. Among the oral manifestations, candidiasis is the most common oral manifestation. The other oral manifestations diagnosed in this study are periodontal disease, recurrent Aphthous ulcers, Melanotic hyperpigmentation, Herpes Zoster infection and fissured tongue. To conclude, oral manifestations are seen in HIV infection and candidiasis is the predominant one. The oral cavity being readily accessible, the oral diagnostician can play an important role in early diagnosis of HIV infection.

The relative incidents of different oral manifestations are given in the Mysore study has 56.25 percent having oral candidiasis. The Table 9.2 shows distribution of other lesions in the mouth.

Table 9.2: Relative incidence of oral lesions in the Mysore study (2000)

Sl No.	*Oral lesions*	*Percentage*
1	Candidiasis	56.25
2	Periodontal disease	25.00
3	RAU	12.50
4	Melanotic Pigmentation	18.75
5	Herpes zoster	6.25
6	Fissured tongue	6.25

Jonsson N et al[12] 1998 studied 100 patients with oral manifestations of AIDS and found that in the Zimbabwean series the median age was 35 years and male to female ratio was 4:1. The systemic symptoms consisted of weight loss in 52, diarrhea in 34, lymphadenopathy in 21 and Herpes zoster in 12 patients. Ninety-two percent patients had oral lesions, which consisted of oral ulcerations in 26 and candidiasis in 22. Neoplasm's recorded were Kaposi's sarcoma in 72 patients, squamous cell carcinoma in two patients. No hairy leukoplakia was found in this study.

Palmer CD et al (1996)[11] studied 456 patients with HIV infection and found that 80 percent of patients with full-blown AIDS had specific oral disease. The most common in their series were Hairy Leukoplakia (30%), Erythematous candidiasis (24%), Psuedomembranous candidiasis (14%), Angular chelitis (6%), Necrotizing periodontal disease (8%) and not specified ulceration (6%).

DIAGNOSIS OF AIDS

AIDS has protean manifestations, which evolve from symptoms termed as ARC (AIDS Related Complex) to generalized lymphadenopathy to generalized wasting and finally to fully blown AIDS.

Tests are classified into:

- Non-specific tests
- Specific tests

Nonspecific Tests

Hematological picture may show

1. Lymphocytopenia (below 2000/ cu. mm)
2. Decrease in CD4 count
3. Low T4 : T8 cell ratio— T helper/T suppressor cells.
4. Increase in IgG and IgA
5. Decreased delayed hypersensitivity on skin testing and deceased natural killer cell activity.

HIV infected person remain seronegative for about 6-12 weeks during "window period" when initial viral replication takes place. Antibody test at this stage does not reveal the true status as it takes some time for formation of antibodies. Therefore a person during this stage will not be aware of infection and capable of transmitting the virus to others.

Specific Tests

They are of two main types:

HIV Antibody Tests and Polymerase Chain Reaction (PCR)

HIV ANTIBODY TESTS

- **Screening test**—ELISA (Enzyme linked immunosorbent assay) is most widely used, inexpensive, good screening test where the HIV infected serum shows positive to antibodies. It has sensitivity of about 99.5 percent
- **Confirmatory tests**—Western Blot is a very useful confirmatory test, where screening test shows positive

results. This test is usually sufficient evidence for proceeding towards a treatment protocol.

- **IFA** (Indirect Immunofluorescent Assay)—This test can detect IgM HIV antibodies very early.
- **RIPA** (Radio-immunoprecipitation Assay)—This is more sensitive and specific than the western blot test. But it is more time consuming and expensive test than the western blot.

Polymerase Chain Reaction (PCR)

- This technique is used for the detection as little as one genome of non-replicating HIV-1 in mononuclear cells. This technique has the capability of detecting latent HIV infection in the non-replicative state in patients who are seronegative.

FDA has approved an EIA (enzyme immunoassay test) developed by Abbot laboratories and recommended it in screening during the window period.[17]

OraSure® is one of the tests for detection of the virus in saliva approved by the FDA. Other tests using saliva are being researched currently.[18]

MANAGEMENT

Use of HAART

Highly Active Anti-retroviral Therapy (HAART)—use a combination of the anti-retroviral drugs like Efavirenz, Nelfinavir, and nucleoside reverse transcriptase inhibitors has considerable caused relief for the HIV patients in the west. This reduces the emergence of drug resistant mutants as compared with the single drug therapy.

However, South and Southeast Asia with its 6 million cases (19.6% of the world cases) and Sub-Saharan Africa—20.8 million cases (68% of the world cases) cannot get the maximum benefit of this therapy due to its expensiveness and non-availability.

What should a Dentist do?

1. All HIV infected individuals develop oral alterations during the course of HIV disease. These oral lesions are readily visible and can be diagnosed early and easily with a clinical examination and good medical history. So dentists shall keep their eyes peeled for the oral manifestation of HIV infection.
2. Take a good sexual history as a part of evaluation of oral mucosal disorders.
3. Ask for preliminary HIV screening tests in dental patients who have tuberculosis, unexplained cervical lymphadenopathy or any nonhealing oral ulcers that is not carcinoma.
4. Healthy HIV infected individuals should be treated in general dental settings which reduces the stigma associated with the disease as well as ensures a certain level of confidentiality.
5. The dental care provider is the most appropriate health care professional who can treat pain and discomfort associated with different oral manifestations in HIV infected patients.
6. To minimize complications after dental procedures while treating HIV infected individuals, dentists should be aware of increased bleeding tendencies, post-operative infections, drug interactions and adverse reactions.
7. Dentist must be a member of the multi-disciplinary team of health care professionals who can administer HAART and other modalities to the patient.
8. Dental professionals should educate other health care colleagues about screening or oral abnormalities in HIV infected patients. This can be accomplished by linking up with regional AIDS education and training centers, giving formal and informal lectures and sending informational updates to health care institutions and community based organizations.
9. Take active part in promoting awareness of AIDS, the role of prostitution and sex workers in spread of AIDS and the importance of clean and protected sex.

Protected sex message will have to be promoted by all the health care workers. It will reduce HIV infection and hopefully it will reduce India's exploding population!!

CONCLUSION

AIDS has been described as the curse of Gods to punish the man who has started sinning, uncontrollably. The psycho-social factors of the promiscuous sexual behavior and intravenous drug abuse have led to medical social workers and research workers to look at factors which are cultural and societal which modify the spread of this disease through the population.

Oral diagnosticians play an important role in early detection of diagnosis of the lesions. The early diagnosis of the lesions can foresee the developments of a suppression of the immune system and can be important as an indicator of the possibility of the early detection of AIDS.

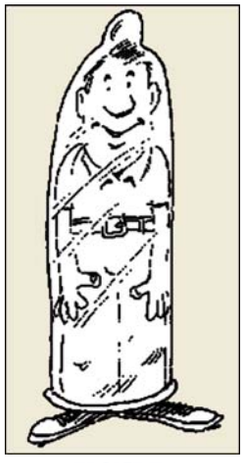

FIGURE 9.4

As scientific understanding about HIV infection has increased, mankind is nearing the doomsday, clearly it is a straight fight between man on one side and the AIDS pandemic on the other. The people of the world need to reach out to each other. There should be no "them" or "us", if this pandemic is to be overcome. It is the time to demonstrate our capacity towards scientific progress. A journey through the dark side of the pandemic. A journey of change, a hope that stigma, fear, and despair are replaced by optimism and compassion.

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Bailoor DN, Chatra LK, Thiruneervannan

10 Facial Pain and Neurological Diseases

INTRODUCTION

Pain is definitely one of the commonest symptoms that drive the person to seek dental care. Pain is also the most written about my both poets, writers and scientific researchers and yet remains poorly elucidated.

DEFINITION

Pain could be defined as a reaction to any obnoxious stimulus, which may herald some kind of underlying pathology. The International Association for the Study of Pain definition is as follows: "An unpleasant sensory and emotional experience normally associated with tissue damage or described in terms of such damage."

Pain is not a simple sensation; it is a complex neurobiological event. Melzack [30] has described that pain has three dimensions (see Fig. 10.1);

- Sensory—Discriminative
- Cognitive—Evaluative
- Affective—Motivational

Pain may be divided into Acute and Chronic. Acute may be one that begins suddenly or it may be of short duration like few hours to a day. The chronic on the other hand, may be one that due to its long-standing and insidious nature (months together) itself becomes a disease. Pain due to chronic Rheumatoid arthritis or Osteoarthritis is a notable example.

THEORIES OF PAIN

A nerve ending which responds to the external stimuli is the nociceptor. Two types of nociceptors have been identified. A-delta high threshold mechanoreceptor, and the C-polymodal nociceptor, which conveys the sensations of mechanical, thermal and chemical stimuli. Many of the advances in study of the response of these nerve fibers has been possible due to many techniques and notable amongst them are the microneurography which were invented by Hagbarth and Vallbo (1968).[26] Nociceptors, which have been previously stimulated, have a property that has been described as sensitization (Fig. 10.2). This sensitization changes the response characteristics of the nociceptors in which both thresholds and latency is reduced. This could be the underlying mechanism for the Hyperalgesia a process in which the tissue which is pathologically changed, or damaged shows lesser tolerance to stimuli.

Okeson JP[31] has quoted Fields who has divided the processing of pain from the Stimulation of nerve fibers to subjective experience of pain in four steps: NP A. Transduction, B. Transmission, C. Modulation, D. Perception.

A. Transduction: It is the stimulation of afferent nerve fibers from noxious stimulus.

B. Transmission: This relates to the process by which peripheral nerves relay information to Central Nervous system.

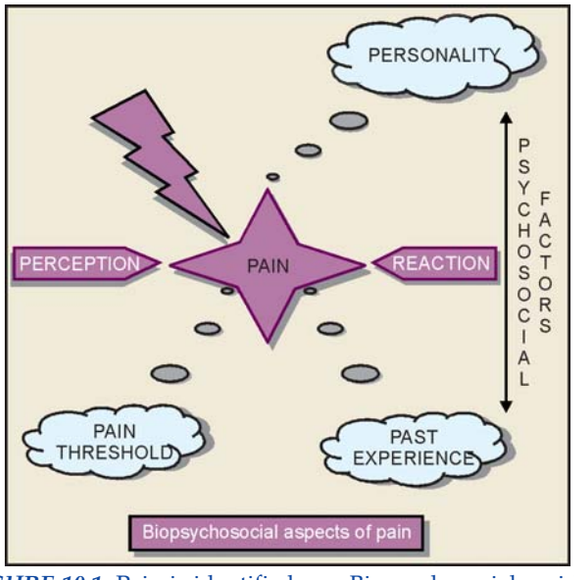

FIGURE 10.1: Pain is identified as a Biopsychosocial variable which is effected by various factors like Intensity of pain stimulus (sensory), persons threshold (cognitive) and modified by past experience and personality factors (affective) (Bailoor DN, Chatra LK 2004)

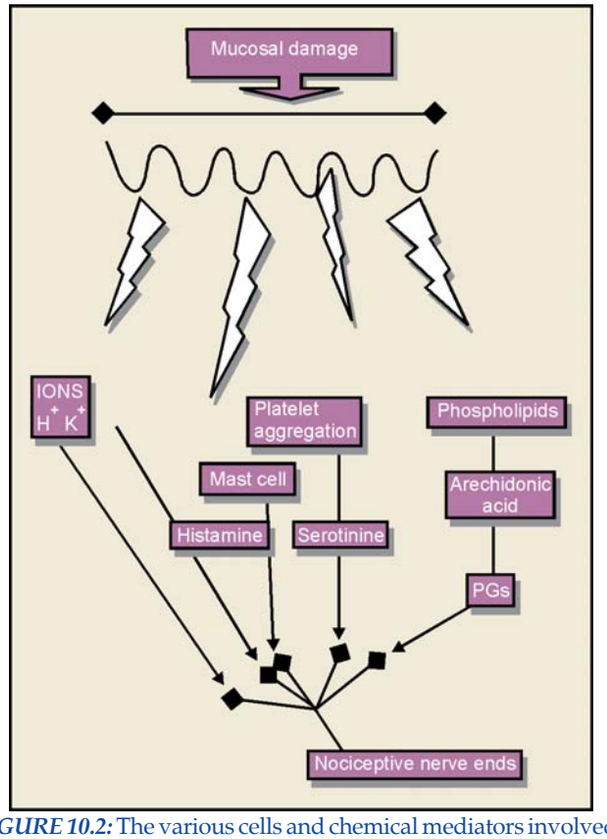

FIGURE 10.2: The various cells and chemical mediators involved in nociception is depicted here (Bailoor DN, Prasanna Kumar, Nillofer Shabnam 2004)

C. Modulation: It is the physiological process that is responsible for the modification of the nociceptive information to the pain centers in the brain. This modification may be done via the neurochemicals specifically norepenephrine, serotonin, Gabapentin which are released by the action of higher centers and provide inhibitory action. The higher centers action results in differing pain thresholds. These thresholds may also be determined by genetic influences or by past learning.

D. Perception: This is the final step in the subjective experience of pain, that is the "Feeling of pain" in the brain. The location has been determined approximately as 'Insula' in the anterior cingulated cortex region. Emotions, learning and past experiences all tend to affect the pain perception.

Two major theories of pain have developed over years.

- The specificity theory of pain and
- The Gate control theory of pain

Broadly the ***specificity theory*** stated that each of the sensations like pain, heat, touch, etc. has specific end-organs, specific neural pathways and unique centers of nociception.

The ***Gate control theory*** proposed by Melzack and Wall (1965)[27] mentions that there is a concept called 'spinal gating mechanism' in dorsal horns which modulates the afferent nerves impulses. The large diameter (L fibers) tend to close the gate [hampers nerve impulses] and the small diameter (S fibers) tend to open the gate [allows the nerve impulses unhampered]. A descending pain control system also controls the amount of transmission and ultimately the perception of the nociceptive stimulus. The descending control of the spinal gate is attributed to the release of powerful neurochemicals termed as Endorphins. (Three types—enkephalins, beta-endorphins and dynorphins)

With the advances in research in nociception both the theories appear to come closer to each other and as a practicing dental surgeon one needs to see these theories in light of clinical applications highlighted later in the chapter.

CLASSIFICATION OF OROFACIAL PAIN

The facial pain should be broadly classified as dental or non-dental. In dental causes rule out the caries, pulpal

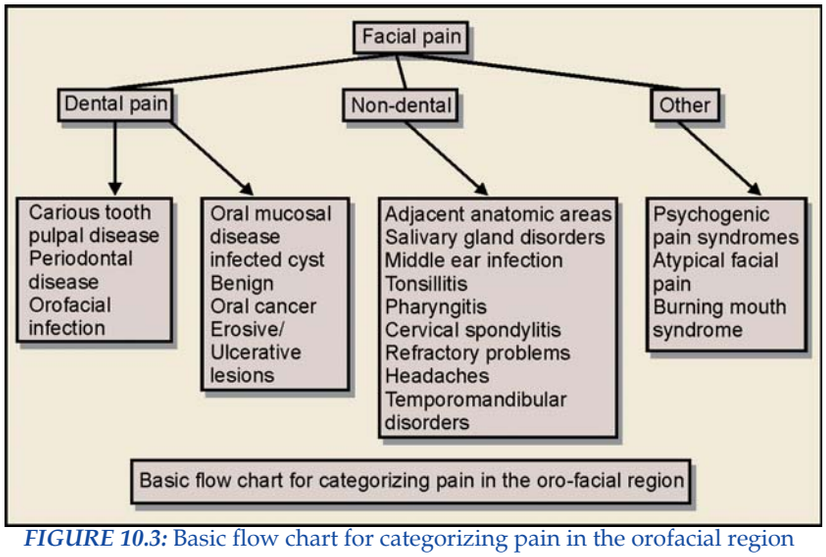

FIGURE 10.3: Basic flow chart for categorizing pain in the orofacial region

diseases, periodontal disease, periapical lesions, and the pericoronal infections. A good OPG, Periapical radiograph of the areas of pain/tenderness is evident clinically and good clinical examination should indicate the cause.

Etiological classification of Facial Pain and related neurological disorders (see Fig. 10.3).

1. **Local causes:** Dentinal lesions, Pulpal lesions, Periapical lesions, Periodontal lesions, Salivary gland disease, Tonsillar diseases, Para nasal sinus disease, middle ear disease, ocular disease, neck—vertebrae related.
2. **Neurological causes:** Trigeminal Neuralgia (Ngia), Glossopharyngeal Ngia, Post Herpetic Ngia, Raeder's Ngia, Intra-cranial disease, Epilepsy, Facial paralysis
3. **Psychogenic causes:** Tension headaches, MPDS, Atypical Facial Pain
4. **Vascular causes:** Migraine, Migrainous Neuralgia and Temporal Arteritis
5. **Other causes** Referred Pain, Raised Intra-cranial pressure, Meningeal irritation, Eagle's syndrome, Drugs (nitrites, dapsone, etc.), Lyme disease.

Pain Control—Methods

1. Analgesics
2. Anti-inflammatory drugs
3. Combination with surgical drainage/extraction and antibiotic therapy (infection related pain)
4. TENS
5. Acupuncture- electro acupuncture
6. Yoga and Meditation
7. Physiotherapy—Hot and hold fomentation, Jaw exercises, etc.
8. *Special methods:* Infrared Lasertherapy, Radiofrequency thermocoagulation, Hypnosis, Stereotactic Gamma knife, Massage therapy

In the following sections only the non-dental aspects of pain will be discussed in detail. The pulpal and periodontal origins of pain have been discussed in other texts.

NEURALGIA

Classification

- Trigeminal Neuralgia TN
- Glossopharyngeal Neuralgia GN
- Herpetic and post-herpetic neuralgia PHN
- Raeder's Paratrigeminal neuralgia RPN
- Neuralgia like pain caused by intracranial lesions NIL

Pain that arises due to injury to the nerves either direct or indirect is referred as Neuralgia. Neuralgic pain is characterized by electric shock like quality, it is limited to the anatomic pathway of the nerve involved rarely crosses midline. It is described as sharp, lancinating, burning or shooting and of unbearable kind by patients.

It can be divided into continuous or paroxysmal. The classical example of paroxysmal neuralgia is Trigeminal neuralgia. The patients of TN normally have an unkempt

look, unshaved, untidy and smelling. Reason is that any of the routine hygiene habits tend to trigger this pain of horrendous proportions. Depending on the branch of the Trigeminal nerve involved. Five to six percent of the patients have bilateral pain episodes and others only one side with only one of the branches being involved. In most cases, no changes are observed in routine radiographic and Histopathologic studies. Mishra B et al (1993)[17] reported three cases of neuritic leprosy mimicking neuralgia. Astute dentist working in leprosy endemic areas of India should keep this in mind for differential diagnosis (see Fig. 10.4).

Vertebrobasilar ectasia has been reported as compressing the fifth cranial nerve in three cases assessed by Kirsch E et al[34] from Basel in 1996 using high resolution MRI.

Neurovascular compression has emerged as the most frequent etiology in cases of Hemi-facial spasm and TN as per a report from Toulouse (France); Holley P et al (1996).[4]

Holley has also mentioned that Magnetic Resonance Angiography (MRA) is the investigation of choice in his assessment schedule. He used this sensitive diagnostic technique with a multi-planar reconstruction program. The neurovascular compression at root entry zone (REZ) has been unequivocally demonstrated in most cases. The neurovascular conflict was related to postero-inferior cerebellar artery (PICA in 61% cases) and anterior-inferior cerebellar artery (AICA in 23% cases).

Janetta PJ (1997) mentions that MRI imaging might reveal in rare instances evidence of Intra-cranial tumor or involvement of nerves in multiple sclerosis.

When Fifty-two patients with facial pain were examined clinically by Ogutcen-Toller M et al.[33] The most frequently observed extracranial pathologic change was sinusitis in their series. They recommend that all the patients who have unexplained pain need to undergo MRI investigation to exclude any intra cranial lesion.

Malignant lymphoma metastasis was the cause of TN in report by Inatomi Y et al (1998)[8] and MRI helped Gass A et al (1997)[35] to localize lesions of multiple sclerosis in his patient suffering from TN but they concluded that in MS trigeminal neuralgia, demyelinating lesions affecting pontine trigeminal pathways were the etiology.

TN is treated by using one of these drugs Tegretol, (Carbamazepine); Dilantin, (phenytoin), Baclofen, (lioresal) and Neurontin, (gabapentin).

Delzell JE Jr et al (1999)[16] mention that there are several promising new medications available, such as pimozide, tizanidine hydrochloride, and topical capsaicin.

In many cases the medications become less and less effective and the dosages have to be increased. This makes the side effects a tangible hazard. In such cases surgical options need to be explored.

Young RF et al (1997)[6] have indicated their successful use of the Leksell Gamma Knife, a sterotactic radiosurgical instrument using 70 Gy dose. They have mentioned a success rate of 81.7 percent who had complete relief thus proving to be safe and effective therapy of TN.

Infarction of the root entry zone of the trigeminal nerve has been reported as a cause by Golby AJ et al (1998)[3] and he mentions that Glycerol Rhizotomy may be indicated as Rx of choice.

Use of Percutaneous Retrogasserian Glycerol Rhizolysis (PRGR) has been evaluated by Eide PK and Stubhaug A (1998)[2] and found to be highly effective in their series of 39 patients.

Tortuous vertebrobasilar artery associated with primitive Trigeminal artery has been mentioned as an etiology by Fukuda et al[7] 1998. Post-surgical Trigeminal neuralgia has been mentioned by Grigoryan YA et al[5] 1999 from Moscow after removal of contra-lateral posterior cranial fossa tumor.

Zakrzewska JM et al[36] utilized McGill Pain Questionnaire (MPQ) and Hospital Anxiety and Depression (HAD) scale and found that many of the TN patients also exhibited Atypical facial pain with severe anxiety and depression. Psychological, sociological and patients' views must be included in evaluations to obtain successful treatment.

Glossopharyngeal Neuralgia (GN)

Sudden shooting pain which seems to be originating from the posterior aspect of the tongue. Swallow is difficult and patient may be afraid to eat anything. Emaciation and nutritional problems may aggravate the depression caused by this kind of paroxysmal pain.

Costantini D et al (1997)[22] in a series of 700 patients treated revealed that electro acupuncture and laser reflex therapy should be considered one of the alternatives for successful therapy of neuralgias.

Post-herpetic Neuralgia (PHN)

The infection of VHZ virus results in pain and vesicles in the anatomic area of distribution of that nerve. The pain usually subsides within a period of three weeks. If the pain persists for more than a month of healing of lesions it may be termed as PHN. This problem is seen more in the elderly patients.

Ernst ME et al (1998)[19] opine that oral corticosteroids are not indicated in control of post herpetic neuralgia and the benefit they confer is limited compared to the risk of dissemination of the viral particles. The presence of hypertension, diabetes mellitus or psychiatric illness further contraindicates the steroid therapy.

Hoffmann V et al (1994)[20] treated a patient of post herpetic neuralgia of the ophthalmic nerve with initially subcutaneous ketamine and then oral ketamine and reported complete recovery. The possible mechanism of action mentioned was by its N-methyl-D-aspartate (NMDA) blocking properties.

Jackson JL et al (1997)[21] have performed a meta-analysis of relevant literature and revealed that treatment of herpes zoster with 800 mg five times a day of oral acyclovir within 72 hours of rash onset may reduce the incidence of residual pain in 6 months by 46 percent in normal adults.

Raeder's Para-trigeminal Neuralgia

Patients complain of unilateral facial pain, constant headaches, vague, undefined with any one of the eyelid drooping and the clinician may observe constriction of pupil. Sore spots are evident in different parts of the skull that may change with time. Sudden, paroxysmal pain, shooting around the orbit region. Ocular sympathetic paralysis and the sudden flashing headaches are probably two symptoms that should alert the clinician. A CT study and MR imaging where available is highly indicated prior to any long-term treatment. Similar medication is indicated as for other neuralgias.

FACIAL NERVE PARALYSIS

The VIIth cranial nerve is the motor nerve of the facial muscles and Viral attacks, stroke or ischemia may result in the facial nerve paralysis. See flow chart below (see Fig. 10.5):

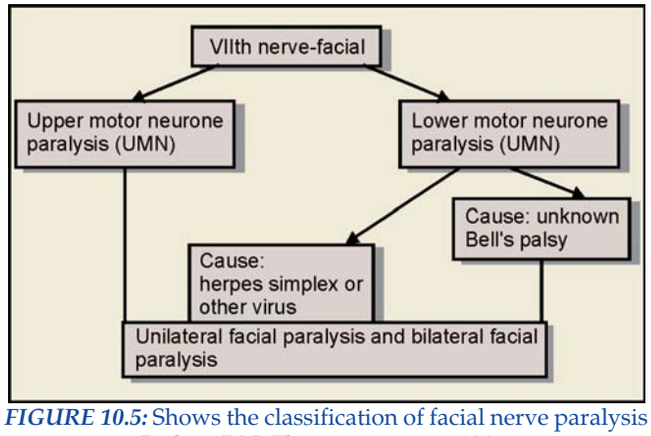

FIGURE 10.5: Shows the classification of facial nerve paralysis Bailoor DN, Thiruneervannan 2004.

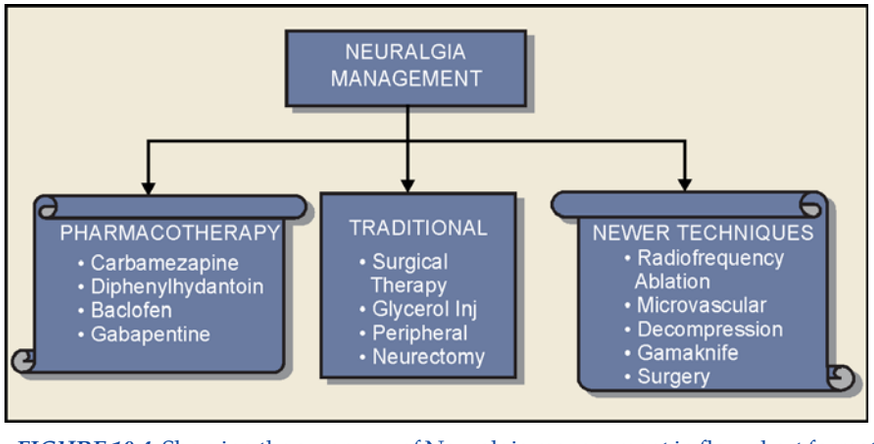

FIGURE 10.4: Showing the summary of Neuralgia management in flow chart format (Prasanna Kumar, Nillofer Shabnam, Bailoor DN 2004. Yenepoya Dental College and Hospital, Mangalore)

Check for five clinical findings— to distinguish Lower motor neuron (LMN) and Upper motor neuron (UMN) lesion.

Characteristic	*Lower motor neuron*	*Upper motor neuron*
On protrusion of tongue	Deviates to unaffected side	No deviation
Blink reflex	Negative	Positive
Wrinkle on forehead	Negative	Positive
Lacrimation and taste	Affected	Unaffected
Closure of eye (Bell's Sign)	Positive	Positive

In two-third of the lower motor neuron facial paralysis no cause is detected and then it is technically termed as Bell's palsy .

Chupryna HM et al (1997)[13] treated 87 patients with multi-modality treatment which included a Biomed-001 unit of infra-red laser used on acupuncture points found significant relief with 890 nm + 20mW intensity. Ongoing research in infrared laser acupuncture should give new directions to this and other neurological disorders.

Bauer CA and Coker NJ (1996)[14] have put forward a hypothesis that Bell's palsy is a herpes simplex neuritis and advocates routine facial nerve decompression for recurrent palsies.

James DG (1997)[18] mentions that when bilateral facial paralysis occurs in young adults the most common cause to be ruled out should be sarcoidosis.

LYME DISEASE

This disease is caused by a spirochaete *Borrelia burgdorferi, Borrelia garinii* etc. This disease has been observed in game wardens, hunters or people coming in contact with deer. An ixodes tick from deer is the culprit carrier. The bite mark pains severely and a reddish spreading macular lesion marks the beginning of the suffering. Knee joints, facial pain and rarely TMD may result. Facial paralysis and lymphadenitis are observed in sporadic cases.

Heir GM (1997)[9] clearly indicate that in perplexing cases of oro-facial pain Lyme disease must be ruled out. Serum antibody levels against the *B. burgdorferi* logically would be increased. CSF analysis is a good factor to check the prognosis. Specific ELISA test is available to diagnose for Lyme disease.

Belman AL et al (1997)[11] in a prospective study of 40 children with-lyme disease associated facial nerve paralysis revealed that WBC count, Protein level or both were abnormal in 68 percent of the cases. Smouha EE et al (1997)[12] in a review of 50 patients with facial nerve paralysis who were seropositive for Lyme disease showed that erythema migrans and meningoencephalitis could not be used to diagnose Lyme disease since it appeared only sporadically. In endemic areas all facial paralysis patients should be evaluated for serological and cerebrospinal tests.

Hashimoto Y et al (1998)[15] used a nested polymerase chain reaction-restriction fragment length polymorphism analysis (nested PCR-RFLP) which was performed on DNA extracted from a skin biopsy of the erythema on the left forehead of a 64-year-old female patient. *Borrelia flagellin* gene DNA was detected and its RFLP pattern indicated that the organism was *B. garinii.* They suggest that nested PCR-RFLP analysis might be useful for the rapid diagnosis of Lyme disease and for evaluating therapy.

The children with borreliosis with or without facial paralysis normally are prescribed penicillin G or ceftriaxone intravenously for at least 14 days. Allergy and related factors may always not make this therapy possible. Alternatively Dotevall L and Hagberg L (1999)[10] in their nonrandomized study of 29 patients treated with oral doxycycline (daily dose, 200-400 mg) for up to two weeks. Ninety percent of the patients showed recovery in 6 months. This is an effective and convenient therapy for Lyme neuroborreliosis.

Graff-Radford SB et al (1995)[28] found that using the electronic thermography may be helpful in differentiating neuropathic type of pain from pulpal pain. The former gives cold thermograms and pulpal pathology gives warm ones.

EPILEPSY

Epilepsy is a disorder of brain function, which may cause loss of consciousness with motor and sensory discharges. Most cases are idiopathic and a few have intra-cranial lesions, febrile episodes or some metabolic disorders that explain the attacks. Primary care giver here is a Neurologist

and dentist has to work very closely with him to take care of his oral health.

Dentist must be ready to treat the injuries to oro-facial complex like tongue lesions, fractures to the mandible or maxilla, TMJ subluxation, avulsion of teeth or rarely swallowing of teeth.

Other aspects of anti-epileptic drugs (AED) are the gingival hyperplasia, folate deficiency symptoms like anemia, recurrent apthous ulcers RAU, dental anomalies, rarely erythema like reactions to the AED.

Newer drugs like flumazenil are being advocated for lesser side effects and management of refractory cases. Sharief MK et al (1993)[23] treated 12 adults with refractory epilepsy, results suggest that oral flumazenil may have an intrinsic antiepileptic activity and provide a rationale for a new approach in the treatment of intractable epilepsy.

Reisner-Keller LA and Pham Z (1995)[24] agrees with above findings and mention that Flumazenil can be added safely to diazepam therapy. Long random controlled studies need to be done before it may be routinely advocated.

Krauss GL et al (1996)[25] comment that Hepatic enzyme-inducing antiepileptic drugs (AEDs) lower oral contraceptive (OC) sex hormone levels approximately 40 percent and increase the risk of unplanned pregnancies in women with epilepsy. AEDs also increase the risk of birth defects in offspring of women with epilepsy. Increasing OC doses can compensate for insufficient OC sex hormone levels due to AEDs dentists being a part of the health care team must know this fact.

In case of Epileptic fit on the dental chair. First of all lift and keep the patient on the floor. Turn his neck so any vomitus or saliva drools out and does not go into his respiratory tract. Any soft paddle may be inserted into the patients mouth to prevent the tongue from being bit. Inject 10 mg diazepam i.m. or 2 mg clonazepam i.m. (slow intravenously may not be practical if convulsions persist). Call in a medical practitioner and check the need to move the patient to a place with nursing and neurological consult.

Oro-dental complications of epilepsy may be various injuries to facial region caused by the epileptic episode or one of the side effects of the drugs prescribed by the physician or neurologist. Drug side effects specifically to be observed are fibrous hyperplasia of gingiva, Folic acid deficiency (look for RAU, and megaloblastic anemia), and sometimes dermal lesions caused by Erythema multiform like lesions by medications administered).

ATYPICAL FACIAL PAIN

It is a vague type of poorly localized pain. Following criteria are recommended before the dental practitioner can brand a patient's pain as atypical pain:

- All the tests and clinical examination should reveal lack of detectable pathology in the dental and oral region, a minimum, dental radiograph, OPG Ortho-pantomograph, biopsy and complete hematologic report should be attempted, together with neurologist's opinion.
- A definite hypochondriac patient who keeps detailed notes about his illness from last many years is more likely candidate.
- A patient who changes his pain location and character every time he reports back.
- When the pain does not follow anatomic pathway, crosses midline and traverses across body planes, its likely to be an atypical facial pain.
- Once diagnosed the patient should be counseled, and depression and anxiety inventory may be administered to determine his psychological status and minor tranquilizers, and antidepressants may be prescribed after consulting with an Oral Medicine expert. If unmanageable then Psychiatrist should be called in. Routine analgesics do not seem to work in this atypical pain syndrome.

SPHENOPALATINE NEURALGIA

It is characterized by unilateral fits of pain in region of eyes, mastoid, zygoma, upper face and nasal area. No trigger zones and comes at the same time each day, hence the term "Alarm clock headache". Spontaneous remissions reported. Treatment-Alcohol injection in the Sphenopalatine Ganglion.

Frey's Syndrome

This syndrome follows the surgery of the parotid or ramus region, this results in damage to the para-

sympathetic nerves which when regenerating result in cross innervation and so whenever the patient has his meal, the temporal area appears to sweat profusely. Treatment is done by surgery intracranially on the auriculotemporal nerve.

Barodontalgia[32]

Once referred to as "Flyer's toothache". Barodontalgia is defined as tooth pain occurring with changes in ambient pressure. It usually occurs in people who fly or dive. It can develop in conjunction with sinusitis, and in teeth experiencing pulpitis after restorative treatment, new and recurrent caries, intra-treatment endodontic symptoms, dental and periodontal cysts, or abscesses. Although the causal process is not well understood, it may be related to pulpal hyperemia, or to gases that are trapped in the teeth following incomplete root canal treatment.

MIGRAINE

This type of pain is characterized by unilateral frontal and temporal pain of throbbing variety, associated with irritablity and nausea. It is commonly seen in women in 20 to 30 age group and more so in the educated women. Pre-menstrual time aggravates this pain. Ergotamine tartarate dose 1-2mg/day. Max 6 mg/day. For Rx.Migranil®, and Amitriptyline—a tricyclic compound for prevention, 100 mg a day.

Drug therapy, biofeedback training, stress reduction, and elimination of certain foods from the diet are the most common methods of preventing and controlling migraine and other vascular headaches. There are two ways to approach the treatment of migraine headache with drugs: prevent the attacks, or relieve symptoms after the headache occurs.

Drugs used to prevent classic and common migraine include Methysergide maleate, which counteracts blood vessel constriction; Propranolol hydrochloride, which stops blood vessel dilation; and Amitriptyline, an antidepressant. Binder WJ (1999)[29], a clinical professor of head and neck surgery suggests that use of nerve paralyzer botulinum toxin type A, called Botox, shows a great promise, is a new direction in migraine relief

Drug Therapy

- Propranolol 100 mg a day
- Flunarin, Flunarizine 5 mg/10 mg tablets prophylaxis
- Daily dose for adults is 10 mg in divided doses
- Inderal, Propranolol 10, 40, 80 mg tabs
- Daily dose is 80-160 mg 2 to 4 times daily.

Eletriptan hydrobromide Relpax from Pfizer® is one of the drugs being currently tried on this pain problem.

Zolmitriptan (Zomig) is available as regular or orally dissolving tablets of 2.5 or 5 mg and has been giving good results.

Research scientists caution, that many of these medications are high risk for people who have angina pectoris, severe hypertension, vascular, liver, or kidney disease.

SUMMARY

Algology has been defined as the science and study of pain phenomena. An algologist is a student, investigator, or practitioner of algology. A competent dentist needs to be a good algologist too. The critical aspect to remember is that dentist is the specialist of the oral cavity and should rule out all the causes of intra oral causes of pain before venturing to call a pain of Atypical facial pain, MPDS, Migraine or Neuralgic etc. A team approach using the help of a physician, a neurologist and even sometimes a clinical psychologist will help in a complete treatment.

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11 Developmental Disturbances of Dental and Facial Structures

INTRODUCTION

It is estimated that 70 percent of the patients with craniofacial anomalies have a definite genetic component in the etiology.[1] Prenatal diagnosis of chromosomal, metabolic and single gene disorders has become a major part of genetic services allowing parents to know whether or not their unborn child is affected. For sake of easy assimilation we classify the disorders affecting orofacial and dental tissues into-

CLASSIFICATION

I. Developmental disturbances in size of teeth
   - Macrodontia
   - Microdontia

II. Developmental disturbances in shape of teeth
   - Fusion
   - Concrescence
   - Dilaceration
   - Talon cusp
   - Dens in dente
   - Dens evaginatus
   - Taurodontism
   - Supernumerary roots

III. Developmental disturbances in number of teeth
   - Supernumerary teeth
   - Predeciduous dentition

IV. Developmental disturbances in structure of teeth
   - Amelogenesis Imperfecta
   - Environmental enamel hypoplasia; may be due to → Congenital syphilis → Hypocalcemia → Birth injuries → Local infection or trauma → Fluoride; mottled enamel → Idiopathic factors
   - Dentinogenesis imperfecta → Dentin dysplasia → Regional odontodysplasia → Dentin hypocalcification

V. Disturbances of growth (eruption) of teeth
   - Premature eruption
   - Delayed eruption
   - Multiple unerupted teeth
   - Embedded and impacted teeth
   - Ankylosed deciduous teeth

VI. Developmental disturbances of the jaws
   - Agnathia
   - Micrognathia
   - Macrognathia
   - Facial hemihypertrophy
   - Hemiatrophy
   - Abnormalities of dental arch relations

VII. Developmental disturbances of the lips and palate
   - Congenital lip and commissural pits and fistula
   - Double lip
   - Cleft lip and cleft palate

- Orofacial digital syndrome
- Cheilitis glandularis
- Cheilitis granulomatosa
- Hereditary intestinal polyposis syndrome
- Labial and oral melanotic macule

VIII. Developmental disturbances of the oral mucosa
- Fordyce's granules
- Focal epithelial hyperplasia
- Peutz-Jeghers syndrome
- Papillon-Lefevre
- Dyskeratosis congenita

IX. Developmental disturbances of the gingiva
- Fibromatosis gingivae
- Retrocuspid papilla

X. Developmental disturbances of the tongue
- Microglossia
- Ankyloglossia
- Cleft tongue
- Fissured tongue
- Median rhomboid glossitis
- Benign migratory glossitis
- Hairy tongue
- Lingual varices

XI. Developmental disturbances of oral lymphoid tissue
- Reactive lymphoid aggregate
- Lymphoid hamartoma
- Angiolymphoid hyperplasia with eosinophils
- Lymphoepithelial cyst

XII. Developmental disturbances of the salivary glands
- Aplasia
- Xerostomia
- Hyperplasia of palatal glands
- Atresia
- Aberrancy

DEVELOPMENTAL DISTURBANCES OF THE JAWS

Agnathia

Agnathia is a rare malformation characterized by the absence of the mandible, microstomia, aplasia or hypoplasia of the tongue, and low-set or medially fused ears. It occurs alone or in combination with a variety of malformations such as holoprosencephaly. Yand SH et al[21] have recommended prenatal sonography at 30-32 weeks of gestation may give a hint at developing problem intra-uterine.

Petryk A, Anderson RM et al[20] hypothesized in their animal studies that twisted gastrulation gene through their action of inhibition of bone morphogenetic proteins may cause forebrain defects and alterations in first branchial arch leading to agnathia.

Schiffer C et al[22] in a study of three fetuses have mentioned that problems occur at embryonic days 22 to 26 for the agnathia —otocephaly complex maldevelopment to occur. The cytogenetic and molecular basis of Karyotypic abnormalities is mentioned as the aberrant gene expression of sonic hedgehog and paired-related homeobox genes.

Micrognathia

Micrognathia literally means a small jaw, and either the maxilla or the mandible may be affected. Many cases of apparent micrognathia are due not to an abnormally small jaw in terms of absolute size, but rather to an abnormal positioning or an abnormal relation of one jaw to the other or to the skull, which produces the illusion of micrognathia.

True *micrognathia* may be classified as either (1) congenital, or (2) acquired. The etiology of the congenital type is unknown, although in many instances it is associated with other congenital abnormalities, including congenital heart disease and the Pierre Robin syndrome.

Although it has been suggested that mouth breathing is a cause of maxillary micrognathia, it is more likely that the micrognathia may be one of the predisposing factors in the mouth breathing; agenesis of the condyles also results in a true mandibular micrognathia.

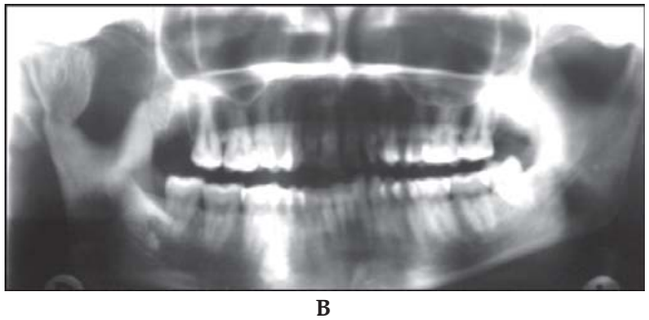
The acquired type of micrognathia is of postnatal origin, and usually results from a disturbance in the area of the temporomandibular joint. Ankylosis of the joint, for example, may be caused by trauma or by infection of the mastoid, of the middle ear, or of the joint itself, since the normal growth of the mandible depends to a considerable extent on normally developing condyles as well as on muscle function.

The clinical appearance of mandibular micrognathia is characterized by severe retrusion of the chin, a steep mandibular angle, and a deficient chin button.

Macrognathia

Macrognathia refers to the condition of abnormally large jaws. An increase in size of both jaws is frequently proportional to a generalized increase in size of the entire skeleton, e.g. in pituitary gigantism. More commonly only the jaws are affected, but macrognathia may be associated with certain other conditions, such as (1) *Paget's disease* of bone in which over growth of the cranium and maxilla or occasionally the mandible occurs; (2) *Acromegaly*, in which there is progressive enlargement of the mandible owing to hyperpituitarism in the adult; or (3) *Leontiasis ossea*, a form of fibrous dysplasia in which there is enlargement of the maxilla.

A



B

FIGURES 11.1A and B: Show OPG radiograph taken in patient complaining of slight asymmetry of face and swelling in preauricular region which moved smoothly on opening and closing mouth. There was no pain or discomfort and radiographic diagnosis of Unilateral condylar hyperplasia was made (Courtesy of Varghese Mani GDC Calicut 2004)

General factors, which conceivably would influence and tend to favor mandibular prognathism are as follows:

- Increased height of the ramus
- Increased mandibular body length
- Increased gonial angle
- Anterior positioning of the glenoid fossa
- Decreased maxillary length
- Posterior positioning of the maxilla in relation to the cranium
- Prominent chin button, and
- Varying soft-tissue contours.

Surgical correction of such cases is feasible. Ostectomy, or resection of a portion of the mandible to decrease its length, is now an established procedure, and the results are usually excellent from both a functional and a cosmetic standpoint.

Facial Hemihypertrophy

A very mild degree of facial asymmetry is present in nearly all persons. Occasionally, however, congenital hemihypertrophy may occur, involving

(1) The entire half of the body
(2) One or both limbs, or
(3) The face, head, and associated structures.

Although the unilateral facial hypertrophy is the most striking feature in patients with this disturbance, it is also the most significant finding to the dentist (see Fig. 11.1).

Etiology

The cause is hypothesized to be:

- Hormonal imbalance
- Incomplete twinning
- Chromosomal abnormalities
- Localized alteration of intrauterine development
- Lymphatic abnormalities
- Vascular abnormalities, and
- Neurogenic abnormalities. Of all of these, the last two seem the most plausible.

Clinical Features

Patients affected by facial hemihypertrophy exhibit enlargement of one half of the head. In some cases this is obvious even at birth. The enlarged side grows at a rate proportional to the uninvolved side, so that the disproportion is maintained throughout life, although

growth of the entire face generally ceases by the age of 20 years. Familial occurrence has been reported. There appears to be some relationship between hemihypertrophy and neoplasms of the kidney, liver, and adrenal cortex in children (Beckwith wiedemann syndrome, etc). Females are affected somewhat more frequently than males (63% versus 37%), and there is almost equal involvement of the right and left sides.

Oral Manifestations

The dentition of the hypertropic side, could be abnormal in three respects:

- Crown size
- Root size and shape, and
- Rate of development.

Characteristically, the permanent teeth on the affected side develop more rapidly and erupt before their counterparts on the uninvolved side. Coincident to this phenomenon is premature shedding of the deciduous teeth.

The tongue is commonly involved by the hemihypertrophy and may show a bizarre picture of enlargement of lingual papillae in addition to the general unilateral enlargement and contralateral displacement. In addition, the buccal mucosa frequently appears velvety and may seem to hang in soft, pendulous folds on the affected side.

Treatment and Prognosis

There is no specific treatment for this condition other than attempts at cosmetic repair.

Differential Diagnosis

There are certain diseases of the jaws, such as neurofibromatosis and fibrous dysplasia of the jaws, that may give the clinical appearance of facial hemihypertrophy, but these can usually be differentiated readily by the lack of effect on tooth size and rate of eruption.

Facial Hemiatrophy

Facial hemiatrophy is a progressive atrophy of some or all of the tissues on one side of the face, occasionally extending to other parts of the body.

Etiology

The causes of the condition suggested are:

- Atrophic malfunction of the cervical sympathetic nervous system
- Trauma infection
- Heredity w peripheral trigeminal neuritis, and
- A form of localized scleroderma.

Clinical Features

The onset of the condition is usually noticed in the first or second decade of life as a white line, furrow, or mark on one side of the face or brow near the midline.

There may be hollowing of the cheek, and the eye may appear depressed in the orbit. The response of the atrophic facial muscle to faradic stimulation may be unaltered. The cartilage of the nose, ear, larynx, and palpebral tarsus also may become involved. In addition, contralateral Jacksonian epilepsy, trigeminal neuralgia, and changes in the eyes and hair commonly occur. Affected skin often becomes darkly pigmented, although vitiligo sometimes develops. Loss of facial hair is common.

Oral Manifestations

Hemiatrophy of the lips and the tongue is reported, as are dental effects. Growth of teeth may be affected just as other tissues are involved.

Treatment and Prognosis

There is no specific treatment for the condition. It has been found that typically the disease will be progressive for a period of several years and then remain unchanged for the remainder of the patient's life.

Abnormalities of Dental Arch Relations

Many different types of malocclusion exist, and many classifications, have been evolved in an attempt to unify methods of treatment. The classification of Angle, proposed in 1899, is the most universally known and used.

Class I: Arches in normal mesio-distal relations as visualized by the first permanent molar relation, or canine relation.

Class II: Mandibular arch distal to normal in its relation to maxillary arch.

Division 1. Bilaterally distal molar relation and protruding maxillary incisors.

Division 2. Bilaterally distal molar relation and retruding maxillary incisors.

Subdivision: Unilaterally distal molar relation

Class III: Mandibular arch mesial to normal in its relation to the maxillary arch.

Subdivision: Unilaterally mesial molar relation.

Since these abnormal jaw relations are studied in detail in the science of Orthodontics, no further discussion will be attempted here.

DEVELOPMENTAL DISTURBANCES OF THE LIPS AND PALATE

Congenital Lip and Commissural Pits and Fistulas

Congenital lip pits and fistulas are malformations of the lips, often following a hereditary pattern, that may occur alone or in association with other developmental anomalies such as various oral clefts. In 3 out of 4 of all cases of congenital labial fistulas, there is an associated cleft lip or cleft palate or both. Commissural pits are an entity probably very closely related to lip pits, but occur at the lip commissures, lateral to the typical lip pits. This entity is frequently hereditary, possibly a dominant characteristic following mendelian pattern, and may be associated with other congenital defects.

Etiology

Many theories of the etiology of congenital lip pits have been offered. Pit may result from pinching of the lip at an early stage of development, with fixation of the tissue at the base of the notch, or from failure of complete union of the embryonic lateral sulci of the lip, which persist and ultimately develop into the typical pits. Commissural pits may occur at the site of the horizontal facial cleft and may represent defective development of this embryonic fissure.

Clinical Features

The lip pit or fistula is a unilateral or bilateral depression or that occurs on the vermillion surface of either lip but far more commonly on the lower lip. In some cases a sparse mucous secretion may exude from the base of this pit. The lip sometimes appears swollen, accentuating the appearance of the pits.

Treatment

Since the pits are harmless and seldom manifest complication no treatment is indicated.

Double Lip

Double lip is an anomaly characterized by a fold of excess tissue on the inner mucosal aspect of the lip. It may be congenital or acquired as a result of trauma to the lip.

Clinical Features

This redundant mass of tissue usually occurs on the upper lip, although the lower lip and, or rare occasions, both upper and lower lips are involved. When the upper lip is tensed, the double lip resembles a "cupid's bow". The double lip usually cannot be seen when the lips are at rest. There is no information available concerning familial, sex, or racial predilection. Occasionally, it occurs in random association with other oral anomalies.

The occurrence of acquired double lip in association with blepharochalsis and nontoxic thyroid enlargement is known as Ascher's syndrome. Blepharochalsis is a drooping of the tissue between the eyebrow and the edge of the upper eyelid so that it hangs loosely over the margin of the eyelid. It is caused by relaxation of the supratarsal fold as result of atrophy and thining of the skin of the eyelid. In these cases, the eye and lip abnormalities usually develop abruptly.

Treatment

No treatment is necessary except for cosmetic purposes or functions involving speech and mastication. The excess tissue is easily excised surgically.

Cleft Lip and Cleft Palate

Facial clefts occur along many planes of the face as a result of faults or defects in development or maturation of

embryonic processes. Thus, we may recognize such anomalies as the oblique and transverse facial clefts, which extend from the upper lip or ala of the nose to the eye and from the angle of the mouth to the ear, respectively. By far the most important of the facial clefts, however, is the cleft lip, mandibular or maxillary. The mandibular cleft lip is an extremely rare condition that occurs in the midline of the lower lip. The more common clefts occur as two separate and distinct entities;

- Cleft lip with or without associated cleft palate, and
- Isolated cleft palate.

The maxillary cleft lip is the more common and important of the lip clefts.

The usual maxillary cleft lip at one time was thought to be due to failure of the globular portion of the median nasal process to unite properly with the lateral nasal and maxillary process. More recently, it has been suggested that this cleft is not due to an actual lack of union of the processes but rather to a failure of mesodermal penetration and the obliteration of the ectodermal grooves separating these mesodermal masses that actually constitute the facial processes. Either the absence or deficiency of these mesodermal masses or their failure to penetrate the ectodermal grooves leads to breakdown of the ectoderm, causing cleft formation. Since penetration occurs between either of the paired lateral mesodermal masses and the single central mesodermal mass, it is obvious that the maxillary cleft may be non-union. Occasionally, however, a portion of the central process is defective or absent, and the resulting cleft does appear in the midline. The cleft palate appears to represent a disturbance in the normal fusion of the palatal shelves; failure to unite due to lack of force, interference by the tongue, or a disparity in the size of the parts involved; the soft palate and uvula do not appear to be formed as a result of fusion of parts but rather as a posterior extension of the palatal processes; thus cleft of these structure is basically an extension of a cleft of the hard palate.

Etiology

Heredity is undoubtedly one of the most important factors to be considered in the etiology of these malformations. However, there is increasing evidence that environmental factors are important as well. Less than 40 percent of the cases of cleft lip with or without cleft palate are genetic in origin, whereas slightly less than 20 percent of the cases of isolated cleft palate appear to be genetically derived. Most investigations indicate that the inheritances pattern in cleft lip with or without cleft palate is different from that in isolated cleft palate. The mode of transmission of the defect is uncertain. It has been pointed out that the possible main modes of transmission are either by a single mutant gene, producing a large effect, or by a number of genes (polygenic inheritance), each producing a small effect, which together create this condition. It should be pointed out that cytogenetic studies have failed to reveal visible alterations in chromosomal morphology of the affected individuals. It is presumed that every individual carries some genetic liability for clefting, but if this is less than the threshold level, there is no cleft. When the individual liabilities of two parents are added together in their offspring, a cleft occurs if the threshold value is exceeded. However, even though this is the most common form of cleft, the threshold value is sufficiently high that it is a low-risk type. The second form of cleft is monogenic or syndromic and is associated with a variety of other congenital anomalies.

Dietary Factors

Although there is insufficient evidence that nutritional disturbances cause cleft palates in human beings, abnormal dietary regimens have caused developmental clefts in animals. Cleft palate has been experimentally produced in newborn rats by feeding diets either deficient or excessive in vitamin A to maternal rats during pregnancy. Riboflavin-deficient diets fed to pregnant rats also have produced offspring with a high incidence of cleft palate. The administration of cortisone to pregnant rabbits has induced similar clefts in their young.

Physiologic, emotional, or traumatic stress may play significant role in the etiology of human cleft palate, since stress induces increased function of the adrenal cortex and secretion of hydrocortisone.

Other possible causes of cleft palate include:

- A defective vascular supply to the area involved;
- A mechanical disturbance in which the size of the tongue may prevent the union of parts;

- Circulating substances, such as alcohol and certain drugs and toxins;
- Infections; and
- Lack of inherent developmental force.

One in 800/1000 births shows changes akin to cleft lip or cleft palate. The incidence of cleft lip, with or without cleft palate, increased with maternal age. Although there is variation in reported incidence in the different studies, the condition is common enough to cause concern.

Clinical Features

The maxillary cleft lip may present a varied clinical picture, depending on the severity of the condition. As the names would indicate, the unilateral cleft lip involves only one side of the lip; the bilateral, both sides of the lip. The latter type has given rise to the term 'harelip', which is now frequently applied to all cleft lips. The incomplete cleft extends for a varying distance toward the nostril and frequently involves the palate as well. The complete cleft extends into the nostril and even more commonly involves the palate. The cleft lip and cleft palate are somewhat more common in boys than in girls, and the lip cleft occurs about three times more frequently on the left side than on the right.

The cleft palate may exhibit wide variation in the degree of severity and the involvement of tissue. The patient with cleft palate and cleft ridge exhibits a large defect in the roof of the palate, with a direct opening into the nasal cavity. This midline defect continues anteriorly to the premaxilla, where it then deviates to either the right or the left. Occasionally, the entire premaxillary portion of bone will be missing, and in such instances, the cleft may appear to be an entirely midline defect. The usual cleft ridge, however, appears in the region between the lateral incisor and cuspid teeth, or it may occur between the maxillary central and Lateral incisors. There is frequently a disturbance in the dental structures in this region, so that teeth may be missing, deformed, displaced, or divided, thus producing supernumerary teeth (Fig. 11.2).

According to a PTI report from Kochi[19] The Indian Medical Association (IMA) had sent a four member team to Kasargod, North Kerala which found enough evidence to link the persistent aerial spraying of endosulphan over cashew plantations and congenital malformations in this area.

The isolated cleft palate is associated with other developmental abnormalities in about 50 percent of the cases. The abnormalities reported are congenital heart disease, polydactylism and syndactylism, hydrocephalus, microcephalus, clubfoot, supernumerary ear, hypospadias, spinabifida, hypertelorism, and mental deficiency. Similar anomalies may occur with cleft lip with or without cleft palate.

FIGURES 11.2A and B: 15-year female patient hailing from Kasargod region reported of spontaneous cleft lip and palate. The parents of this patient have been working on cashew farm which was involved in endosulphan spraying about ten years back. Endosulphan is a organoclorine pesticide which was being used for protection of crops by aerial spraying. The link between this pesticide and respiratory illnesses, malignancies and birth defects is being investigated by IMA (Prasanna K, Nillofer S,Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

Cleft lip is usually repaired at one month of age; the cleft palate operation is done at 18 months of age.

Role of Dental Surgeon

- In diagnosis of the condition
- In making a transitional prosthetic plate for the baby to suckle the mother's milk till the surgery is done.
- To assist in the closure surgery
- To diagnose and treat the ensuing malocclusion in the maxillary arch due to surgical contraction of middle face.
- To liaise with psychologist and the speech therapist for the overall improvement in the socialization of patient.

OROFACIAL DIGITAL SYNDROME

Anneren G et al (1984)[10] have described two varieties of this syndrome. Now many authors accept following four types of this syndrome.

1. ***Papillon-Leage syndrome associated with cleft lip or palate*:** These patients present with deficiency of upper lip and nose, which is deformed. Hypertrophied frenum of upper and lower lips. Tongue is multilobed. The teeth are widely separated and in bizarre shapes. Early caries is noted in most teeth.
2. ***Mohr syndrome inherited as recessive trait*:** Broad bifid nasal tip, hamartomas of the tongue, bifid uvula and cleft palate are some features seen in this syndrome.
3. ***Associated with mental retardation and eye abnormalities:*** Mental retardation is seen predominantly. All the problems mentioned above may be seen variably. The digits of hands and feet may be more in number and often fused with each other. Kyphosis and short sternum are seen in most cases.
4. ***Associated with tibial dysplasia*:** Oral manifestations similar to Papillon-Leage syndrome. The changes in tibia due to irregular mineralization is the differentiating point from other three types.

Hairy Tongue

On the tongue elongation of filiform papillae on the dorsum results in a peculiar condition called a hairy tongue. Depending on whether there are keratin and candidal deposits (white hairy tongue) due to yellow pigmentation of tobacco abuse resulting in yellow hairy tongue and long term antibiotic use and radiation therapy resulting in black hairy tongue. The treatment for this condition is symptomatic and assurance should be given to the patient about its harmlessness. A soft brush needs to be used for maintenance of oral hygiene.

True hairs are quite rare in oral mucosa and few rare references are made to this fact. One of them is work by Humeniuk HM et al[9] 1986 who mention of an ectodermal anomaly causing hair in the mucosa.

Andersson G et al[12] 1997 found that increasing consumption of nicotine was directly related to increased average prevalences for leukoedema, smoker's palate and hairy tongue.

Cheilitis Glandularis

The lower lip is swollen and Hyperplastic and slowly becomes everted. Seen rarely, and when it occurs, common in males. Cause unknown. Simple type, superficial suppurative type and the deep suppurative type. Weir and Johnson[8] have reported this to be premalignant lesion in which 18-35 percent of these lesions undergo change to epidermoid carcinoma. Surgical stripping should be attempted and plastic-cosmetic surgeon should be involved in the esthetic reconstruction of the repaired lip.

Cheilitis Granulomatosa (CG)

Diffuse swelling seen on the lips especially lower, the swelling is soft and no pitting on pressure. The Melkerson-Rosenthal syndrome classically depicts the facial paralysis, and scrotal tongue with CG. Cause is unknown and seen both in children and adults.

Labial and Oral Melanotic Macule (OMM)

The labial melanotic macule is a melanotic lesion of the lips which may occur at any age and presents as a single or multiple small, flat, brown or brown-black asymptomatic lesion of the lip, almost invariably

- Post-traumatic pigmentation
- True ephelides

Gingiva, Buccal mucosa and palate has these lesions which are within 1cm in diameter. It is important to

distinguish between the lesions caused by racial pigmentation, endocrinal disturbances, antimalarial therapy Peutz-Jeghers syndrome, trauma, hemochromatosis, and chronic lung disease. OMM is benign and only if there is a sudden change in its character or ulceration should it be biopsied. It is best left alone.

Fordyces Granules

It is a developmental problem of the oral mucosa characterized by a collection of sebaceous glands which are out of place for the oral mucosa. Clinically it varies from small yellow white spots to rarely big bilaterally symmetrical patches of white yellow areas present from birth.

Clinical Features

Fordyce's granules appear as small yellow spots, either discretely separated or forming relatively large (Plaques) often projecting slightly above the surface of the tissue. They are found most frequently in a bilaterally symmetrical pattern on the mucosa of the cheeks opposite the molar teeth but also occur on the inner surfaces of the lips, in the retromolar region lateral to the anterior facial pillar, and occasionally on the tongue, gingiva, frenum, and palate. Ectopic sebaceous glands have been recognized to occur, besides the oral cavity, in the esophagus, the female genitalia including the cervix uteri, the male genitalia, the nipples, the palms and soles, the parotid gland, the larynx, and the orbit.

Fewer children than adults exhibit Fordyce's granules, probably because the sebaceous glands and hair system do not reach maximal development until puberty.

Treatment

These glands are innocuous, have no clinical or functional significance, and require no treatment. However, very rarely a benign sebaceous gland adenoma or a keratin filled pseudocyst may develop from these intraoral structures.

Focal Epithelial Hyperplasia (Heck's Disease)

The term "focal epithelial hyperplasia" was used to designate a type of lesion first observed by Heck on the oral mucosa of a group of Navajo Indian children. Single or multiple papular lesions which are flat topped, or with multiple filiform projections rarely becoming like papillomatous in appearance. Benign and require no treatment.

Peutz-Jeghers Syndrome

This is characterized by pigmented spots on lips and perioral region. Patients complain of intestinal problems like chronic constipation and pain. Endoscopic biopsy reveals gastrointestinal polyposis. Melena, anemia and prolapsed rectal polyps are mostly seen. Diagnosis is based on barium studies and endoscopic biopsy. Endoscopic surgical removal is sometimes warranted in severe cases. Dentist is the primary health care person responsible for early diagnosis.

Oral Manifestations

On the lips and oral mucosa round, ovoid or irregular macules of bluish gray pigment of variable intensity may be seen. The facial pigmentation tends to fade later in life, although the mucosal pigmentation persists. Intestinal polyposis and the chances of increased risk of malignancy of the large intenstine makes barium meal and rectal colonoscopy a part of basic diagnostic work up.

Papillon-Lefevre Syndrome

Here the patients have changes seen in palms and soles, which are called hyperkeratosis. The radiographs and clinical examination show severe destruction of alveolar bone, which is not proportional to local factors. The palm and soles are normally affected from 5 years onwards. The treatment is best left in hands of periodontal specialists.

Rault S et al (1997)[15] have reported an association of filiform palmo-plantar hyperkeratosis with a digestive adenocarcinoma and polycystic kidney disease.

Indira D et al (1999)[13] have commented that usually palms and soles are considered immune to leprosy, but in their study of 280 leprosy patients they observed palmo-plantar lesions in about 10% of the patients screened. This point is well worth remembering for dentists practicing in the leprosy endemic areas in India.

Olivé A et al (1999)[14] have shared their experience of 16 cases of SAPHO syndrome [Synovitis-Acne-Palmoplantar pustulosis-Hyperostosis-Osteitis]. This syndrome was characterized by the severe thoracic pain, sacro-iliac synovitis, and palmo-plantar pustulosis. HLA B27 was negative in all the cases.

Dyskeratosis Congenita (DC)

Patients of this syndrome show hyper pigmentation of the skin, destruction of the nails, oral leukokeratosis and blepharitis. This is inherited probably as autosomal recessive trait. Oral lesions start around 5 years of age and consist of multiple blister lesions. They break down and recur until progressive healing leads to multiple white lesions and may be diagnosed clinically as leukoplakias. Wormer R et al (1983)[11] have given two classical cases and mention that squamous cell carcinoma may be seen at an early age in these leukoplakias. Various treatments including local retinoids and systemic retinoids are being tried.

Sölder B et al (1998)[16] have described DC as an X-linked disorder affecting many of the systems. They opine that the dermatologic and the mucosal changes are but minor compared to the hematologic and immunologic alterations. Pancytopenia seems to be a finding which does affect the prognosis of the case. The precise pathogenesis is still obscure and beneficial effect has been observed by the administration of hematopoietic growth factors (G-CSF, GM-CSF).

Baselga E (1998)[17] have reported the DC with aplastic anemia. Reticulated hyperpigmentation of the neck, upper chest and proximal parts of the limbs are the characteristic findings in these patients. Hyper pigmentation was more pronounced in along the Blaschko's lines.

In summary the DC or Dyskeratosis congenita is a rare, hereditary, multisystem disorder characterized by mucocutaneous changes, pancytopenia and increased incidence of malignancy and lung disease.

Fibromatosus Gingivae (Elephantiasis Gingivae, Hereditary Gingival Fibromatosis; Congenital Macrogingivae)

Fibromatosis gingivae is a diffuse fibrous overgrowth of the gingival tissues. It is transmitted through a dominant autosomal gene. Familial history is usually positive. Hypertrichosis has been associated with some of these cases.

Clinical Features

This is manifested as a dense, diffuse, smooth, or nodular overgrowth of the gingival tissues of one or both arches, usually appearing about the time of eruption of the permanent incisors. The tissue is of normal or even pale color, and it is often so firm and dense that it may prevent the normal eruption of teeth. It is not painful and shows no tendency for hemorrhage. The extent of the tissue overgrowth may be such that the crowns of the teeth are nearly hidden even though they are fully erupted with respect to the alveolar bone.

A definitive history must rule out any drug ingestion which may be related to gingival overgrowth. Seymour RA et al (2000)[18] have discussed the factors affecting the drug induced gingival enlargement in detail.

Treatment and Prognosis

Impedance of tooth eruption warrants surgical removal of the excessive tissue and exposure of the teeth. The cosmetic appearance may also require surgical excision.

Retrocuspid Papilla

The retrocuspid papilla is a small elevated nodule located on the lingual mucosa of the mandibular cuspids.

Clinical Features

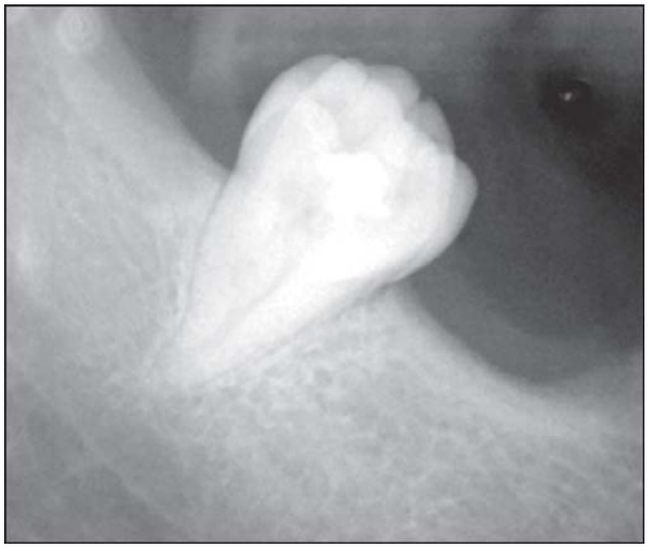
This soft, well-circumscribed, sessile, mucosal nodule, commonly bilateral, is located lingual to the mandibular cuspid, between the free gingival margin and the mucogingival junction. It is exceedingly common in children, occurring in 99 percent of those between the ages of 8 and 16 years and slightly more common in the females. It has to be recognized as a soft tissue landmark.

DEVELOPMENTAL DISORDERS AFFECTING THE TEETH

Developmental Disturbances in Size of Teeth

Microdontia

Teeth which are anatomically smaller than normal are termed as microdonts. It may be localized or generalized.



FIGURES 11.3A and B: Showing 22-year-old male who had come with a chief complaint of carious teeth who also had fusion of a supernumerary tooth to second molar (Prasanna K, Nillofer S, Bailoor DN 2003. Yenepoya Dental College and Hospital, Mangalore)

Localized microdontia involving a single tooth is rather common in lateral incisors. It appears as peg shaped tooth with short roots.

Generalized microdontia: In persons where the jaw inherited is large-the apparently normal teeth also appear small. This may be termed as pseudomicrodontia. Pituitary Dwarfism: Teeth are well formed but over all small in dimensions.

Treatment: In localized a full cover crown appears to improve aesthetics. Similar classification could be done in Macrodontia. No treatment is normally necessary or recommended.

Developmental Disturbances in Shape of Teeth (Fig. 11.6)

Gemination

Geminated teeth are anomalies which arise from an attempt at division of a single tooth germ by an invagination, with resultant incomplete formation of two teeth. The structure is usually one with two completely or incompletely separated crowns that have single root and root canal. It is seen both in the deciduous and permanent.

The term 'twinning' has sometimes been used to disignate the production of equivalent structures by division resulting in one normal and one supernumerary tooth.

Fusion

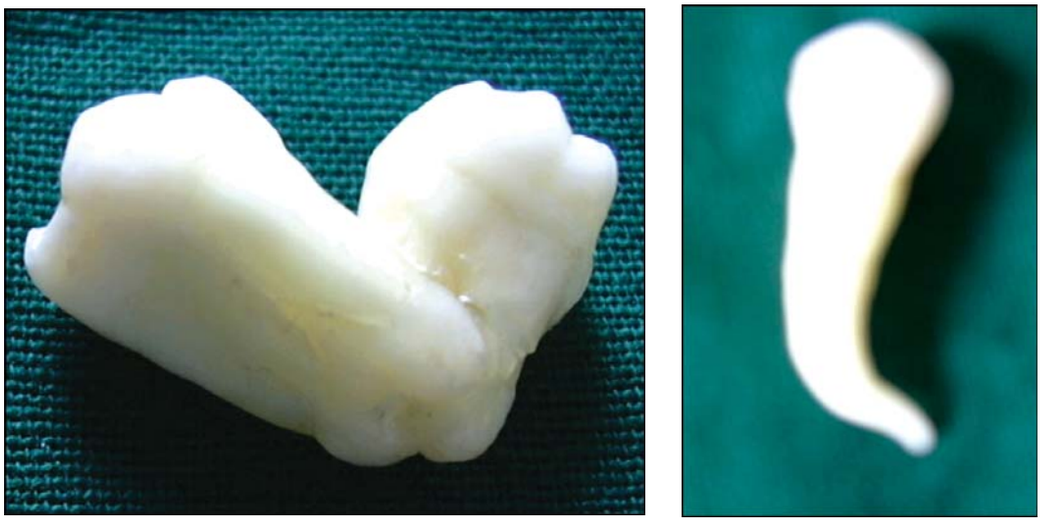
Fused teeth arise through union of two normally separated tooth germs. Depending upon the stage of development of the teeth at the time of the union, fusion may be either complete or incomplete. It is reported that this condition of teeth is more common in the deciduous. Heredity appears to be a principal factor discerned (Fig. 11.3).

Concrescene

Concrescene of teeth is actually a form of fusion which occurs after root formation has been completed. In this condition, the teeth are united by cementum only. The diagnosis can be established by IOPA examination. Since the extraction of one may result in the extraction of the other it is best to recognize this condition prior to any surgical intervention (Fig. 11.4A).

Dilaceration

The term "dilaceration" refers to an angulation, or a sharp bend or curve, in the root or crown of a formed tooth. The curve or bend may occur anywhere along the length of the tooth. Sometimes at the cervical portion, at other times



A. Concrescence **B. Dilaceration**

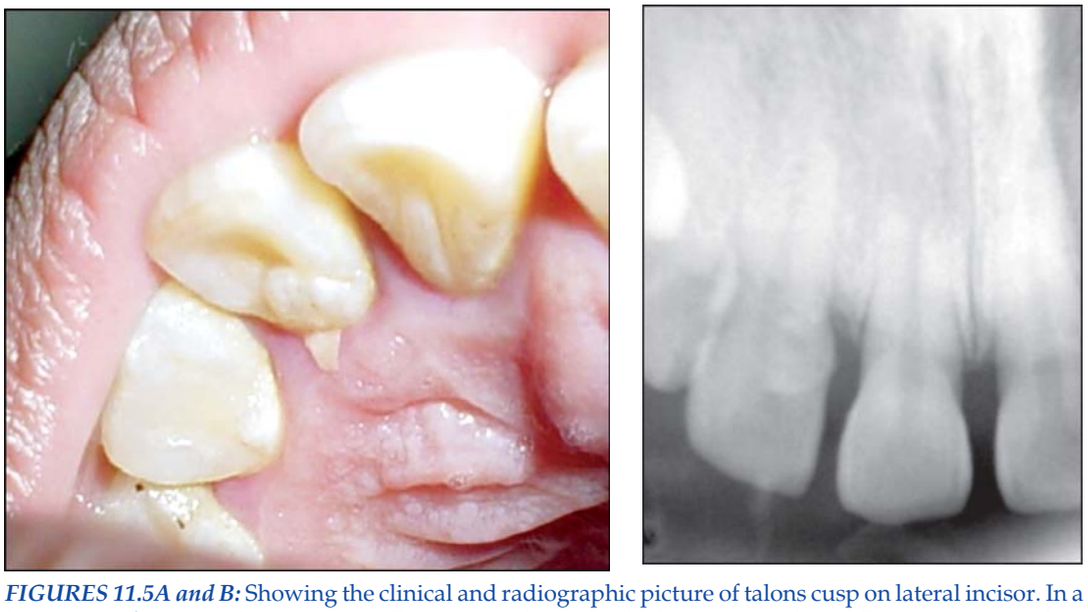
FIGURES 11.4A and B: Specimen photographs of concrescence and dilacerations of root

midway along the root or even just at the apex of the root. The need for preoperative roentgenograms before any surgical procedures are mandatory (Fig. 11.4B).

Talon Cusp

The talon cusp, an anomalous structure resembling an eagle's talon, projects lingually from the cingulum areas of a maxillary or mandibular permanent incisor. This cusp blends smoothly with the tooth except that there is a deep developmental groove where the cusp blends with the sloping lingual tooth surface. It is composed of normal enamel and dentin and contains a horn of pulp tissue.

The *Rubinstein-Taybi syndrome* is associated with developmental retardation, broad thumbs and great toes, characteristic facial features, delayed or incomplete descent



FIGURES 11.5A and B: Showing the clinical and radiographic picture of talons cusp on lateral incisor. In a 17-year-old female patient (Beena K, Omal PM, Bailoor DN 2003 Yenepoya Dental College and Hospital, Mangalore)

of testes in males, and stature, head circumference, and bone age below average (Fig. 11.5).

Dens in Dente (DID)

DID is a morphological aberration which occurs when there is an invagination in the surface of the tooth crown prior to the calcification. Pressure differentials occurring around the tooth germ could probably explain this process. It normally occurs bialterally but it sometimes occurs in the root areas of the teeth. Bhatt and Dholakia[2] explain that this may be due to infolding of Hertwigs sheath and takes its origin within the root after its origin is complete.

Lingual pit area shows a deep invagination which may be missed clinically but radiographically an oval invagination of enamel and dentin with narrow constriction at the opening of the surface. This invagination frequently involves upto the root apex. This is one of the conditions that can be diagnosed with ease with a radiographic examination. Endodontics appears to give uniform results. When this interferes with the occlusion there is a need for endodontic intervention coupled with Occlussal rehabilitation.

Dens Evaginatus

It is developmental aberration that appears as an extra cusp or a small hemisphere of enamel on the occlusal surface. It commonly occurs on premolars between the lingual and buccal cusps. Rarely seen on other teeth. Increased prevalence in Chinese, Japanese, Philippines, Eskimos and American Indians. Whenever such a condition is seen by clinician it is best to radiographically evaluate and treat endodontically. Kumar[4] has presented all the salient features of this disorder for use of the dental surgeon.

Taurodontism (T)

This peculiar anomaly results when the crown of the tooth appear to grow at the expense of the root.

May be classified in various ways one of them being – *Hypo, Meso and Hyper Taurodont.*[1]

These teeth are compared to those of cud chewing animals in their morphology. Mutation and Atavistic feature is said to be some of the causes for this condition. It may be caused by the failure of Hertwig's epithelial sheath to invaginate at the proper horizontal level. Neanderthal man was said to have teeth similar to these. Klinefelter syndrome also is said to show a differing frequency of T. Permanent teeth are more commonly affected.

Radiographically T is seen as a rectangular teeth, increased apico-occlusal dimensions, pulp lacks the constriction at neck of tooth region. The furcation may normally be few mm from the apex of the tooth. No special treatment is necessary.

Supernumerary Roots

It is common to see extra roots in mandibular bicuspids and cuspids. This highlights the routine necessity of IOPA radiographs prior to all extractional procedures.

Anodontia—Supernumerary Teeth—Predecious Dentition—Postpermanent Dentition

Anodontia or absence of teeth could be total involving all the teeth and partial involving some of the teeth.

Developmental Disturbances in Number of Teeth

Total anodontia may be seen in Hereditary Ectodemal Dysplasia. Partial anodontia in which one or more of the teeth appear to be missing, commonest being third molar (35%) Deciduous teeth missing is uncommon— but may occur in maxillary lateral incisor, mandibular lateral incisor and mandibular cuspids. The cause appears to be genetic and in many instances families have shown similar teeth being missing (Fig. 11.9).

Supernumerary Teeth: Gardener's Syndrome

It commonly presents with extra teeth termed as supernumerary teeth. Fader and Associates.[3] This syndrome consists of (Figs 11.7 and 11.8):

a. Multiple polyposis of the large intenstine
b. Osteomas
c. Many epidermoid or sebaceous cysts of the skin (scalp and back)
d. Impacted supernumerary teeth.

These are extra teeth, which may develop from the additional tooth bud or from the split in the permanent tooth bud. Common site is the midline of maxilla between

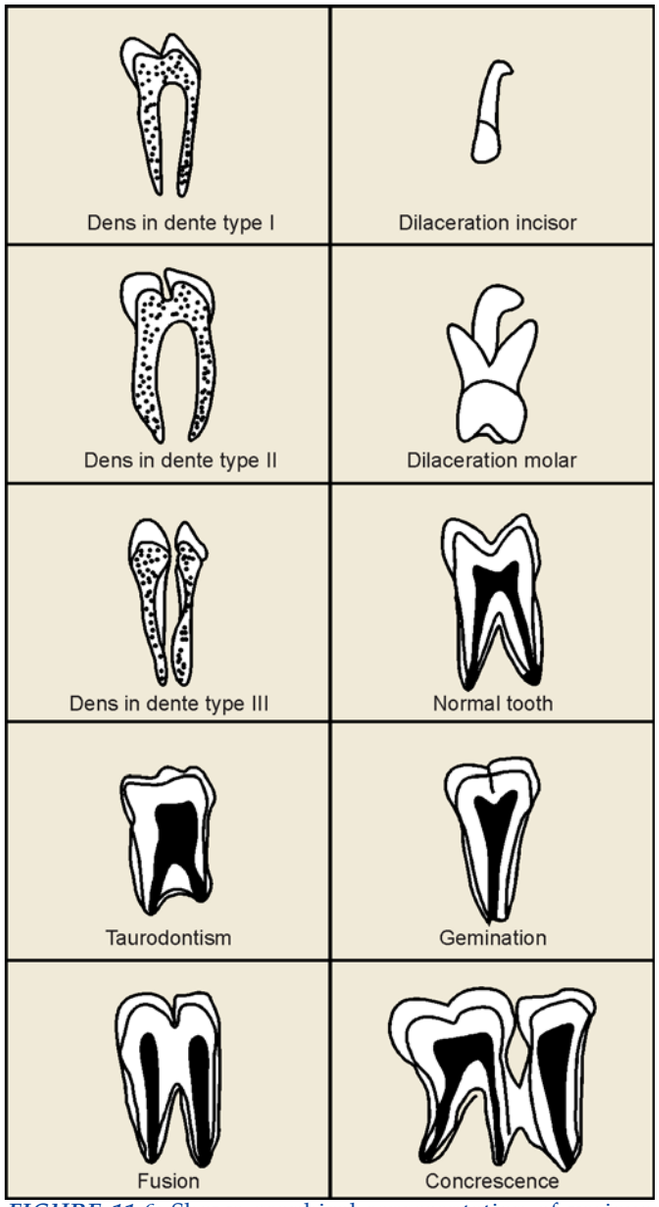
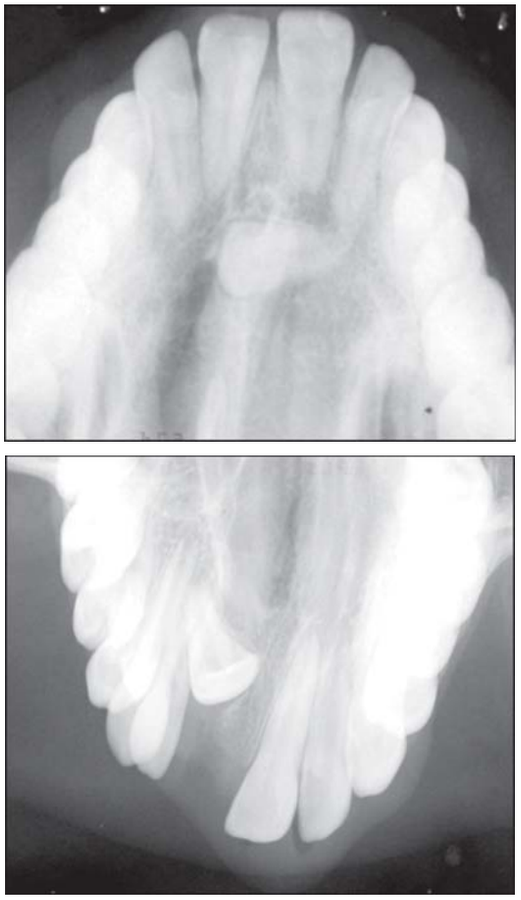


FIGURE 11.6: Shows graphical representation of various developmental disturbance of tooth. Nillofer S, Prasanna K, Bailoor DN 2004.

the two central incisors termed as the 'mesiodens'. It is normally a short cone shaped tooth either erupted, impacted in various locations in midline of maxilla.

Distomolar is the next type supernumerary seen distal to the third molar also often called as the fourth molar. Those extra molars that occur either buccally or palatally to existing molars are termed as paramolars. In the deciduous tooth maxillary lateral incisor has been mentioned in the supernumerary series.



FIGURES 11.7A and B: Figure showing impacted teeth on occlusal radiograph (a) shows an impacted canine which is placed horizontally (b) shows a unerrupted and vertically impacted central incisor which due to its oblong positioning appears to have a dilacerated crown. (Omal PM, Beena K, Bailoor DN 2003 Yenepoya Dental College and Hospital, Mangalore)

FIGURE 11.8: Shows supernumerary teeth present in the midline, commonly referred as mesiodence. Which has cause crowding and malocclusion (Beena K, Omal PM, Bailoor DN 2003 Yenepoya Dental College and Hospital, Mangalore)

FIGURES 11.9A TO D: 17 year old female patient with chief complaint of missing teeth and esthetic problems. No history of any extraction performed till date. Maternal history of chickenpox was reported. Menstrual irregularity was being treated for by her physician. She was also on anti-depressant therapy. OPG confirmed the clinical absence of these teeth. Diagnosis: True partial anodontia (Beena K, Omal PM, Bailoor DN 2003 Yenepoya Dental College and Hospital, Mangalore)

Multiple impacted supernumerary teeth are seen in the Cleidocranial Dysostosis and sometimes even in absence of any abnormalities of the clavicle.[5]

Predecidious Dentition

Neonates are born with teeth like structures in the mandibular incisor area. These have been described as hornified epithelial structures without roots. They need to be removed since they may hurt the mother's nipple as the child suckles.

Post permanent dentition arising from the third set of teeth i.e. after the primary and permanent is probably quite rare, and when a complete denture patient in India presents himself with more teeth either erupting or submerged it would probably be just supernumerary impacted teeth missed during the initial preprosthetic examination.

A REVIEW OF GENETIC DISORDERS OF OROFACIAL REGION

P. RAMACHANDRA REDDI

Genetic abnormalities of craniofacial growth can be divided into two broad categories.

- Those resulting from defects in neural crest cell formation.
- Those resulting from defects in neural crest cell migration.

Deficiencies in the initial number of neural crest cells are frequently reflected in aberrant development in midfacial derivatives and are usually accompanied by defects in the forebrain and ocular structures. The second group of anomalies that results from an apparent hindrance of normal crest cell migration may be involved

in the genesis of the midline cleft malformations, which is characterized by severe orbital hypertelorism. This group of malformations frequently referred to as fronto-nasal dysplaisa, rarely have accompanying brain abnormalities or mental retardation.

Some of the examples of the indirect effects of gene mutation on craniofacial development are conditions like achondroplasia and other chondrodystrophies that produce characteristic facial deformities by virtue of their effect on chondrocranial growth. The neurocranium is particularly susceptible to a number of genetic defects ranging from chromosomal to endocrine in etiology. Defects of facial bones may occur as part of various genetic disorders. Among the more noteworthy anomalies are the scooped out facial appearance due to maxillary hypoplasia and a depressed nasal bridge in achondroplasia.

The genetic disorders affecting the orofacial structures may be divided into those affecting the various structures of the teeth and those syndromes with oral manifestations.

HEREDITARY DEFECTS OF THE ENAMEL

Some gene mutations affecting the structure or composition of enamel usually result in alterations detectable only in the enamel, others may also involve alterations in other tissues or metabolic processes. In general, these mutations result in one of the following

- Insufficient enamel being formed (hypoplasia).
- A marked defect of initial calcification of the organic matrix (hypocalcification).
- A defect in the formation of crystalline apatite in various components of the enamel rods of enamel sheaths (hypomaturation).

Present evidence suggests that a minimum of one X-Chromosomal and three autosomal foci are involved in non-syndromal defects in enamel.

Amelogenesis Imperfecta

Hypoplastic Amelogenesis Imperfecta

The hypoplastic spacing forms of amelogenesis imperfecta include those disorders in which all or a localized portion of the enamel does not reach normal thickness during development. In general, these conditions appear clinically as a thin enamel on teeth that do not contact each other mesio distally, as pits in the enamel or as horizontal fissures in the enamel.

Hypocalcified Amelogenesis Imperfecta

The enamel is so soft, that it may be lost soon after eruption, leaving a crown composed of only dentin. The enamel has cheesy consistency and the enamel at the cervical portion of the crown is often better calcified than that on other portions of the crown. Numerous teeth may fail to erupt and have a marked delay in eruption.

Radiographically, the teeth show short roots with sharp, apical constrictions. Multiple periapical radiolucencies, are of great value in making the diagnosis of dentin dysplasia. Obliteration of the pulp is a significant feature of this disorder, and root canals are absent. Defective dentin is also seen in some syndromes like Vitamin D- resistant rickets, Albrights hereditary osteodystrophy.

Dentinogenesis Imperfecta

This is a disorder of laying down of dentine which is usually expressed as autosomal dominant triat. It is also known as hereditary opalescent dentine because clinically teeth appear discolored. DI has been divided into three types (Fig. 11.10):

- **Type 1**—Associated with Osteogenesis imperfecta. And the deciduous teeth are no severely affected.
- **Type 2**—Only dentine is affected and there are no bone changes.
- **Type 3**—Also termed as Brandywine type which has only tooth changes but no bone changes.

Radiographically type 1 and 2 exhibit identical changes like short roots, opacification of pulp chambers and bell shaped crowns. In type 3, however pulp chambers and root canals are extremely large and giving the appearance of shell like teeth.

Dentinal dysplasia is another similar condition which has variable opacification of pulpal tissue but radiographically a typical horizontal radiolucency indicates some residual pulpal tissue.

Odontodysplasia

This abnormality involves both enamel and dentine concurrently leading to the term Ghost teeth. These teeth

FIGURES 11.10A to C: Showing 16-year-old female with chief complaint of teeth chipping and breaking on taking regular food. Clinical examination and radiograph reveals bell like crowns, short roots and opacification of pulp chambers. A tentative diagnosis of Dentinogenesis imperfecta Type 2 was made (Courtesy Ani John, Umarji H GDC Mumbai 2004)

due to poor calcification and retarded morphological development reflect as wispy shadows on the radiographs. Such teeth usually needs to be extracted and full mouth rehabilitation done.

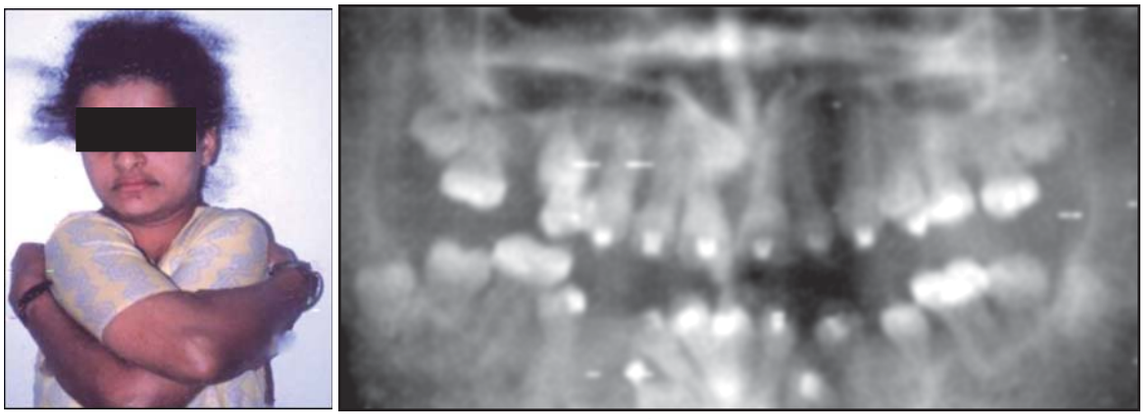
HEREDITARY DISORDERS AFFECTING THE ENTIRE MORPHOLOGY OF THE TOOTH

i. **Taurodontism:** It is discussed earlier in this chapter.
ii. **Odontodysplasia or Ghost like teeth:** Here the calcification of the entire enamel, dentin and cementum appears to look like a ghost, i.e. a pale image of bizarre shape.

Hereditary Disorders of the Periodontal Ligament

Familial Juvenile Periodontosis

It occurs in the adolescent period of life, and females have been reported to be affected more frequently than males. The first clinical evidence is deep pocket formation accompanied by sudden symmetric pathologic drifting of



FIGURES 11.11A and B: Shows clinical and radiographic picture of a patient with Cleidocranial Dysplasia. (a) The clinical photograph shows close approximation of the upper arms due to the absence of the clavicles. Frontal bossing can also be appreciated. (B) The OPG shows multiple impacted teeth which are being corrected orthodontically (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

FIGURES 11.12A to C: Cleidocranial Dysostosis with multiple supernumerary unerupted teeth and lack of collar bone—clavicle bilaterally (Courtesy Ani John, Umarji H, GDC Mumbai)

teeth. Migration is followed by extrusion of the teeth from the socket.

Papillon-Lefevre Syndrome

The principal signs are a) Hyperkeratosis of the palms and soles b) Premature destruction of the periodontal ligament of both deciduous and permanent teeth. Destruction of the periodontal ligament is followed by mobility of the teeth and eventually they are shed.

Cleidocranial Dysplasia

It consists of aplasia or hypoplasia of one or both clavicles, exaggerated development of the transverse diameter of the cranium, delayed dental eruption and supernumerary teeth. It follows an autosomal dominant mode of transmission (Figs 11.11 and 11.12).

The failure of eruption of the deciduous and permanent teeth results in pseudo anodontia. Supernumerary teeth are common. The jawbones may possibly have increased density, which may inhibit tooth eruption.

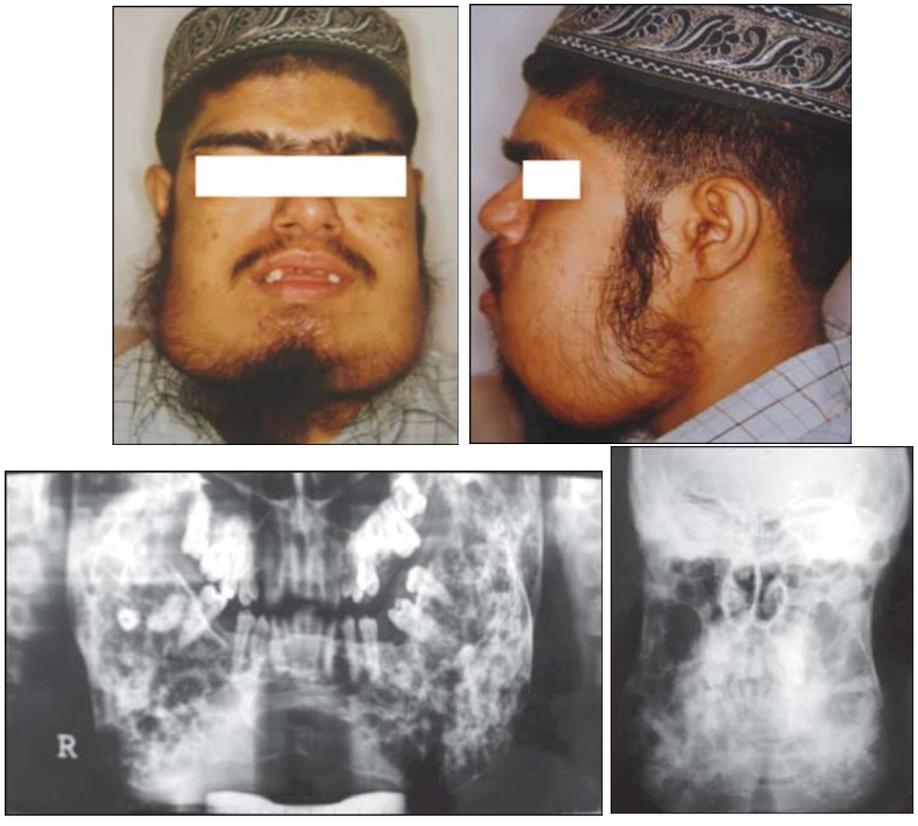
Cherubism

It is transmitted as an autosomal dominant trait with variable expressively. It manifests itself in early childhood. The deciduous dentition may be shed spontaneously and prematurely, beginning as early as 3 years of age. The permanent dentition is often defective, with absence of numerous teeth and displacement and lack of eruption of those present (Fig. 11.13).

SYNDROMES INVOLVING ORO-FACIAL STRUCTURES

Apert's Syndrome

It is a rare variant among the craniostenosis characterized by (a) Oxycephaly (b) Syndactyly of hands and feet.



FIGURES 11.13A to D: Showing painless bilateral swelling of the mandible of over 12 years duration. Patient was only concerned about esthetics. Radiographic appearance was characteristic for Cherubism. The entire trabeculation of the ascending ramus and body has been replaced by radiopaque radiolucent lesion with irregular trabeculations (Courtesy: Ani John, Hemant Umarji GDC, Mumbai 2003)

This syndrome seems to be transmitted by an autosomal dominant gene. The middle third of the face is flat and underdeveloped, producing a relative prognathism. The nose is small and parrot beak shaped. Orbits are flattened with bilateral proptosis.

Oral Manifestations

Abnormalities in the formation of hard or soft palate in the form of a high arched palate, with a marked median furrow. Bifid uvula is also seen. Crowding of the teeth leads to marked thickening of the alveolar process. Rai and Munshi.[6]

Craniofacial Dysostosis (Crouzon's Disease)

It is characterized by a variety of cranial deformities, facial malformations, eye changes and associated abnormalities. It is transmitted as an autosomal dominant trait. The facial malformations consist of hypoplasia of the maxilla, with mandibular prognathism and a high-arched palate. The nose is parrot beak-shaped and the eyes show hypertelorism, exophthalmoses with divergent strabismus and optic neuritis.

Down's Syndrome (Trisomy 21 Syndrome)

It is the most common chromosomal abnormality to occur in man. Patients with Down's syndrome are characterized by a flat face, a large anterior fontanelle, open sutures, small slanting eyes, and open mouth, frequent prognathism.

Oral Manifestations

Macroglossia, with protrusion of the tongue, as well as fissured tongue or pebbly tongue from enlargement of the papillae is seen. The teeth are sometimes, malformed, enamel hypoplasia and, microdontia being most common.

Ectodermal Dysplasia (Anhidrotic or hypohidrotic)

It is characterized by a congenital dysplasia of one or more ectodermal structures and their accessory appendages. It is characterized by a triad of hypodontia, hypotrichosis and anhydrosis. It has an X-linked recessive Mendelian character, males being affected much more frequently than females. Patients exhibit a soft, smooth, thin, dry skin with complete or partial absence of sweat glands. The bridge of the nose is depressed, the supra-orbital ridges and frontal bones are pronounced and the lips are protuberant.

Oral Manifestations

Anodontia with frequent malformation of any teeth present is seen. Since the alveolar process does not develop in the absence of teeth, there is reduction from the normal vertical dimension, resulting in the protuberant lips.

Ehlers-Danlos Syndrome

The characteristic features are the hyperelasticity of skin, hyper extensibility of the joints, fragility of the skin and blood vessels resulting in excessive bruising as well as effective healing of skin wounds.

Oral Manifestations

The oral mucosa is excessively fragile and easily bruised. The gingiva is said to be more liable to injury and periodontal disease occurs at an early age. Radiographically, the teeth may have stunted and deformed roots and large pulp stones in the coronal part of the pulp chamber.

Familial Gingival Fibromatosis

It is manifested as a dense, diffuse, smooth or nodular overgrowth of the gingival tissues, usually appearing about the time of eruption of the permanent incisors. The extent of the tissue overgrowth may be such that the crowns of the teeth are nearly hidden even though they are fully erupted with respect to the alveolar bone.

The combination of gingival fibromatosis, hypertrichosis, mental deficiency and/or epilepsy is genetically heterogeneous with autosomal dominant and recessive types.

Mandibulofacial Dysostosis (Treacher Collins Syndrome)

The important clinical manifestations of this disorder are:

- Anti mongoloid palpebral fissures with a coloboma of the outer portion of the lower lids.
- Hypoplasia of the facial bones, especially the malar bones and mandible.
- Malformation of the external ear

- Macrostomia, high arched palate, abnormal position and malocclusion of the teeth. The characteristic facies, has often been described as fishlike.

Acanthosis Nigricans

The skin lesions appear as dark, velvety hyperkeratotic areas most commonly encountered in intertriginous areas and body fields, such as axillae, umbilicus, and neck.

Oral Manifestations

The tongue and lips are most frequently involved. The tongue shows elongation of the filiform papillae. In addition, papillomatous lesions may be present on the dorsal surface that may resemble verruca vulgaris.

White Sponge Nevus

It follows hereditary pattern as an autosomal dominant trait but with irregular penetrance and no sex predilection. The oral lesions may be widespread, often involving the cheeks, palate, gingiva, floor of the mouth and tongue. The mucosa appears thickened and folded with a soft or spongy texture and a peculiar white, opalescent lure.

MANAGEMENT OF GENETIC DISEASES

One of the most important aspects of management of genetic disease is genetic counseling. Most cases involve studying the specific family pedigree to provide the patient and his family with information regarding the probability of recurrence within that family, as well as the prognosis of the affected individual.[7]

Some techniques like chorionic villus sampling (CVS), which can produce the information about abnormal fetus at ten weeks, are now being advocated in high-risk couples. Doctors also use a blood test known as maternal serum alfa-fetoprotein (MSAFP). This test, usually done between the fifteenth and twentieth week, can detect a neural tube defect of the spinal cord or brain, such as spina bifida or Down's syndrome.

The newest procedure is called BABI (blastomere analysis before implantation). Using reproductive technologies, a couple can conceive several embryos in test tubes and discard those exhibiting known defects. A doctor gives a woman a drug to stimulate ovulation, then extracts eggs from her ovaries and mixes them with her husband's sperm. So far, the procedure has been used to test embryos for such hereditary diseases as Tay-Sachs and Duchenne muscular dystrophy. Thus, genetic counseling can often raise ethical questions, and this is especially true when abortion is involved on detection of abnormalities. Victor Maojo et al[23] have mentioned that the new field of biomedical informatics (BMI) holds great promise for developing informatics methods that will be crucial in the development of genomic medicine. Today the genetic counselors around the world are utilizing blood samples from different patients and trying to determine which of the patients have chromosomal abnormalities and gene level abnormalities. C Wright et al[24] have recommended that Genetic register services incorporating long term follow up and a proactive approach to at risk subjects be attempted scientifically. This would provide an excellent tool of improving access to genetic counseling for families with dominant or X linked genetic disorders and chromosome translocations. In India the doctors should also educate the youngsters against the cultural practice of marrying within the family since this will increase the risk of congenital problems of various types.

SUMMARY

It is not uncommon to find developmental disturbances affecting the orofacial region. Unless associated with systemic problems, they do not necessitate special consideration in treatment.Well-established information on simple genetic control of specific traits, in addition to knowledge of gene frequency within a population, is often adequate to provide a basis for counseling. It is best for most of the practicing dental surgeons to refer the patient's parents to specialized genetic counselors to ensure that next born has a little better chance at normal life.

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12 White Lesions of Oral Mucosa

INTRODUCTION

A practicing dental surgeon in India is commonly confronted by a plethora of white lesions, which can confound the uninitiated new practitioner. The correct knowledge of the clinical features and the confidence to plan the treatment effectively will judge the clinical success of the management of these white lesions.

Mechanisms Why the Lesions Appear White?

Normally the mucosa appears coral pink due to reflection of the light by the underlying capillary bed, when the translucency of the mucosa is lost due to any of the underlying factors the mucosa may appear white (Fig. 12.1).

- Increased thickness of epidermal covering with increased production of Keratin-Frictional Keratosis and Leukoplakia
- Production of abnormal keratin and imbibition of fluids by the upper layer of mucosa—Leukoedema
- Foreign body. Infection, lodging of infection adherent to the superficial layers of the mucosa like. Candidiasis
- Increased fibrosis in the connective tissue and decreased vasculature

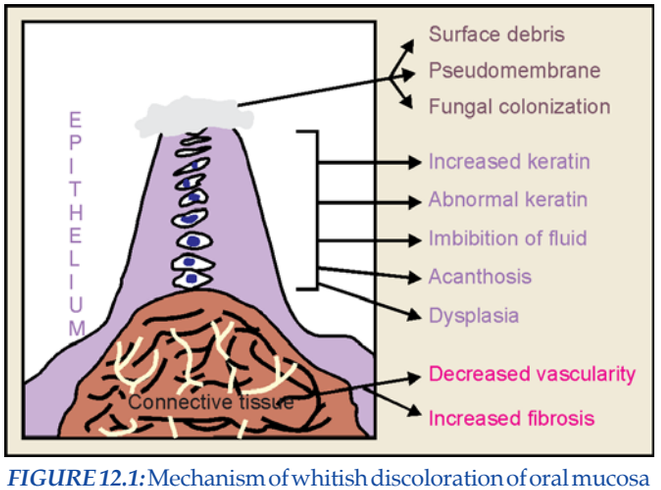


FIGURE 12.1: Mechanism of whitish discoloration of oral mucosa

Classification

First Classification

Clinical basis of keratotic and non-keratotic.

A. Keratotic white lesions (Non-scrapable)

Common:

- Leukoedema
- Linea alba buccalis
- Leukoplakia
- Benign migratory glossitis
- Peripheral scar tissue
- Lichen planus

- Electrogalvanic current lesion
- Lichenoid drug reactions
- White hairy tongue
- Oral submucous fibrosis
- Papilloma
- Verrucous carcinoma
- White sponge nevus
- Skin graft

Rarities:

- Bohn's nodule
- Condyloma latum
- Koplik's spots
- Psoriasis
- Interstitial syphilis
- Hereditary benign intraepithelial dyskeratosis (Seen in some parts of US)—HBID

B. *Sloughing pseudomembranous necrotic white lesions (Scrapable)*

Common:

- Plaque
- Traumatic ulcer
- Pyogenic granuloma
- ANUG
- Candidiasis
- Necrotic ulcers
- Cancrum oris—Noma
- Erosive lichen planus

Rarities:

- Diphtheria
- Eosinophilic granuloma
- Addison's disease

Second Classification

Based on Therapeutic options

White lesions needing urgent/immediate treatment	*Others*
Leukoplakia with indurated, ulcerated edges	Fordyce's granules
Speckled Leukoplakia	Leukoedema
All Leukoplakia on floor of the mouth and ventral surface of the tongue	Linea alba buccalis
Erosive lichen planus Syphilitic mucous patches Oral submucous fibrosis Verrucous carcinomas	HBID

Third Classification

I. ***Variation in structure and appearance of the normal oral mucosa...***
   a. Leukoedema
   b. Fordyces granules
   c. Linea alba buccalis
   d. Frictional proliferation.

II. ***Non-keratotic white lesion.***
   a. Habitual cheek bite
   b. Burns—thermal, chemical (aspirin, dental medicaments) other iatorogenic causes
   c. Radiation mucositis
   d. Uraemic stomatitis
   e. Caused by specific infection
   f. Koplik's spots
   g. Syphilitic mucous patches.

III. ***Candidiasis***

IV. ***Keratotic lesions with no increased potential for carcinoma***
   a. Traumatic keratosis
   b. Oral genodermatoses
   c. Psoriasiform lesions
   d. Intraoral skin grafts

V. ***White lesions with precancerous potential***
   a. Oral submucous fibrosis
   b. Carcinoma in situ
   c. Syphilitic glossitis
   d. Ulcerated leukoplakia
   e. Speckled leukoplakia
   f. Erosive lichen planus
   g. Sideropaenic dysphagia
   h. Stomatitis Nicotina (Fig. 12.3)

VARIANTS OF NORMAL FORDYCES GRANULES, LINEA ALBA, LEUKOEDEMA, HBID—(HEREDITARY BENIGN INTRAEPITHELIAL DYSKERATOSIS)

Fordyce's Granules

Clusters of ectopic sebaceous glands appear as whitish, yellowish plaques, or globular areas, which are bilaterally symmetrical, they are there from birth or they may become

FIGURE 12.2: Showing multiple dispersed whitish elevation on the buccal mucosa, characteristic of Fordyce's granules. This is the common finding and does not have any clinical significance (Nillofer S, Prasanna K, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

more prominent at adolescence. Seen frequently on buccal mucosa, or vermillion border of lips (Fig. 12.2). No treatment is required, only correct identification and assurance of the patient.

Linea Alba

It is a white linear lesion of cheek bite, which occurs at the occlusal line. Sharp cusps of the premolars and molars may be modified. This line is accentuated in people with bruxism, in such cases, causes of anxiety may be identified.

Leukoedema

A generalized grayish white opalescence is imparted to the buccal mucosa by the leukoedema, commonly seen in smokers. More common in Indian Males than females. This opalescence vanishes on stretching. It is not premalignant and so no treatment is needed. Sandstead and Lowe[1] did not find any correlation with the use of tobacco, pH of saliva, oral bacterial infection or galvanic irritation, so it is fairly safe to say that it may be one of the variants of the oral mucosa.

Leukoplakia

Definition

It is a whitish patch or plaque that cannot be characterized clinically or pathologically as any other disease and which is not associated with any physical or chemical causative agent except the use of tobacco.[3]

Risk Factors

The concept of direct etiology in multi-factorial diseases is not accepted today. In its place we see the concept of risk factors emerging in the clinical medicine. These are the factors, which will increase statistically, the chance of that individual getting a particular disease.

Risk factors associated with Leukoplakia are:

1. ***Tobacco Abuse***
   - Chewing
   - Smoking
   - Inhalation/Snuff

   Culturally in India chewing is done with mixture of Betel Nut, Cathechu, and Lime. TLAB (Tobacco+ Lime+ Arecanut+ Betel leaf)

   Smoking cigarettes is an urban phenomenon and in the villages *bidi* smoking predominates, with *hukka* or clay pipe and reverse smoking is seen in some villages of Andhra Pradesh. It is associated with the palatal leukoplakia and even ulcerative changes leading to squamous cell carcinoma.

   Inhalation and snuff are more likely to cause damage to the nasal mucosa and the irritation to maxillary sinus.
2. ***Alcohol abuse:*** Three types of alcohols have been classified in the Indian context .

   The locally made Arrack, Patta and Tharra, which is brewed out of noxious constituents is very strong and even potentially poisonous. The distillery made liquors like rum, gin, brandy, etc. that are distributed legally. And third variety the light liquors like wines, beers, etc.

   In India we find a very strong association between the locally made brews and the occurrence of oral leukoplakia, and even malignancies. This may be due

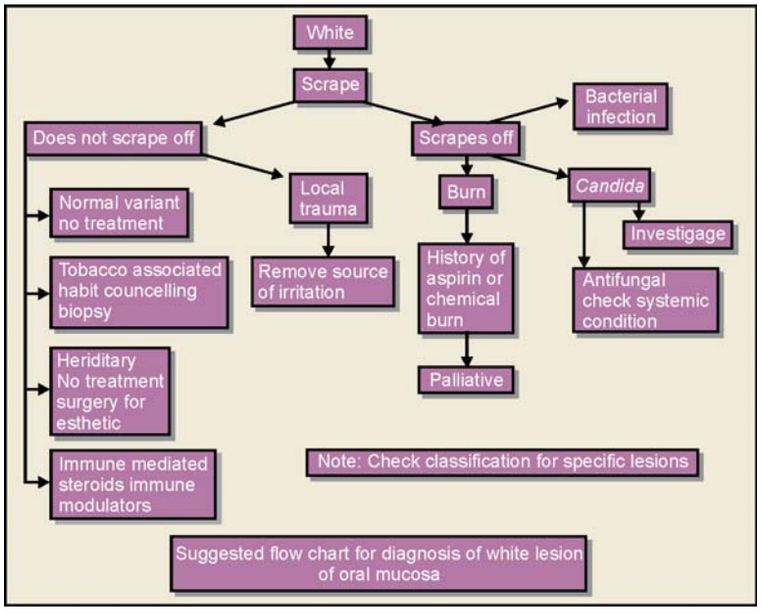


FIGURE 12.3: Suggested flow chart for diagnosis of white lesion of oral mucosa

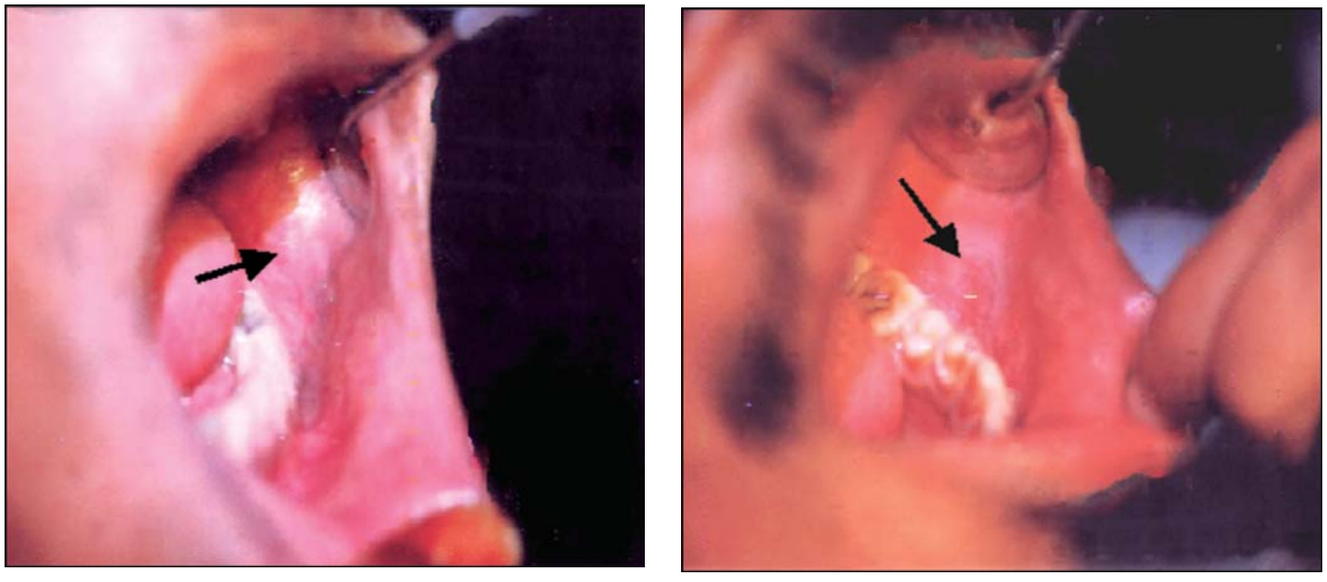
to other factors like low socio-economic status, and malnutrition that predominates the people who indulge in this habit. Some wines have been associated with floor of the mouth Leukoplakias. As a rule most of people who consume alcohol also have concomitant tobacco abuse.

3. ***Vitamin deficiencies:*** Vitamin A and Vitamin B complex especially B_{12} and Folic acid deficiency induces metaplasia and keratinization.
4. ***Actinic radiation:*** Lesions are especially seen in the farmers who work in the fields in South India, in the hot sun of the equatorial region.
5. ***Spices:*** The high amount of spices and chilly used in the Indian subcontinent has raised a number of speculative theories about its role in etiology of leukoplakia but it is not clearly established.
6. ***Local:*** Irritation in form of a sharp denture, prosthetic clasp or sharp edge of a broken carious tooth can aggravate the existing leukoplakia to become ulcerative but its role in primary risk is improbable.
7. ***Syphilis*** is seen less frequently than before in India with the advent of antibiotics. Its lesions are less seen in their full-blown status. However, syphilitic glossitis still remains the classical precancerous oral lesion.
8. ***Viruses:*** Herpes virus hominis type 1 and Human papilloma virus[19] have both seen associated with the development of leukoplakia. Lehner[8] mentions that the malignant transformation of the leukoplakia is a result of failure of cell mediated immune response to the virus.
9. ***AIDS*** has now come to stay in India and as its scourge spreads to give a multitude of oral lesions in which the hairy leukoplakia is one that can be diagnosed by the observant dental surgeon. Wagh and Ani J[27], Mohan.[28] Hairy Luekoplakia does not appear to have any pre-malignant potential.
10. ***Candida:*** Since Candida organisms are commensals in the oral cavity it seems far fetched that the close histopathological association should be construed as etiologic. *Candida* on leukoplakia is also referred as *candidal leukoplakia*.

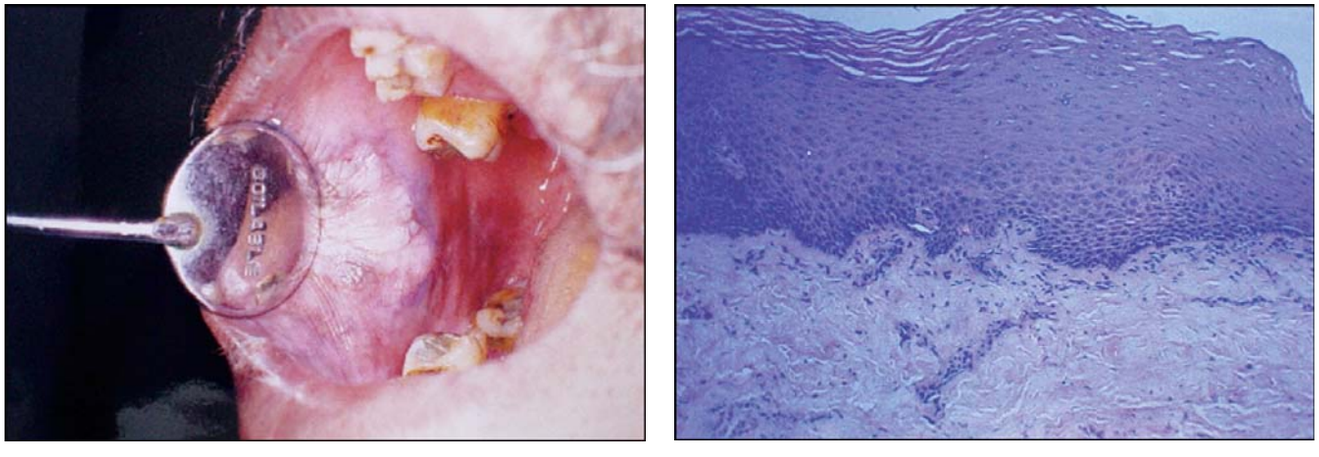
Table 12.1: Showing prevalence of Leukoplakia in Indian subcontinent

No. of persons examined	*Reference*	*Prevalence*
6000	Lay et al (1982)[4]	1.7%
54499	Bhonsle et al (1976)[5]	1.6%
57518	Smith et al (1975)[6]	11.7%
101761	Mehta et al (1972)[7]	0.7%

Average prevalence in this subcontinent appears to be approx 5.5 percent study of Lay et al is from Myanmar a country adjacent to India.



FIGURES 12.4A and B: Showing 18-year-old male patient complaining of burning sensation in the mouth, with a history of pan masala chewing since 5 years. He had a habit of keeping the quid in the buccal sulcus. The mucosa shows whitish discoloration with shriveled appearance in the area where the quid was kept and the lesion merges with the normal mucosa with the indistinct borders. This lesion has been named differently by different authors such as pan chewers mucosa, snuff dippers lesion, preleukoplakia. (Nillofer S, Prasanna K, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)



FIGURES 12.5A and B: Homogenous Leukoplakia in a 45 -year-old male with betel nut chewing, bidi smoking since 22 years. Biopsy revealed mild dysplastic changes (Ani John, Umarji H GDC Mumbai 2004)

Clinical Types

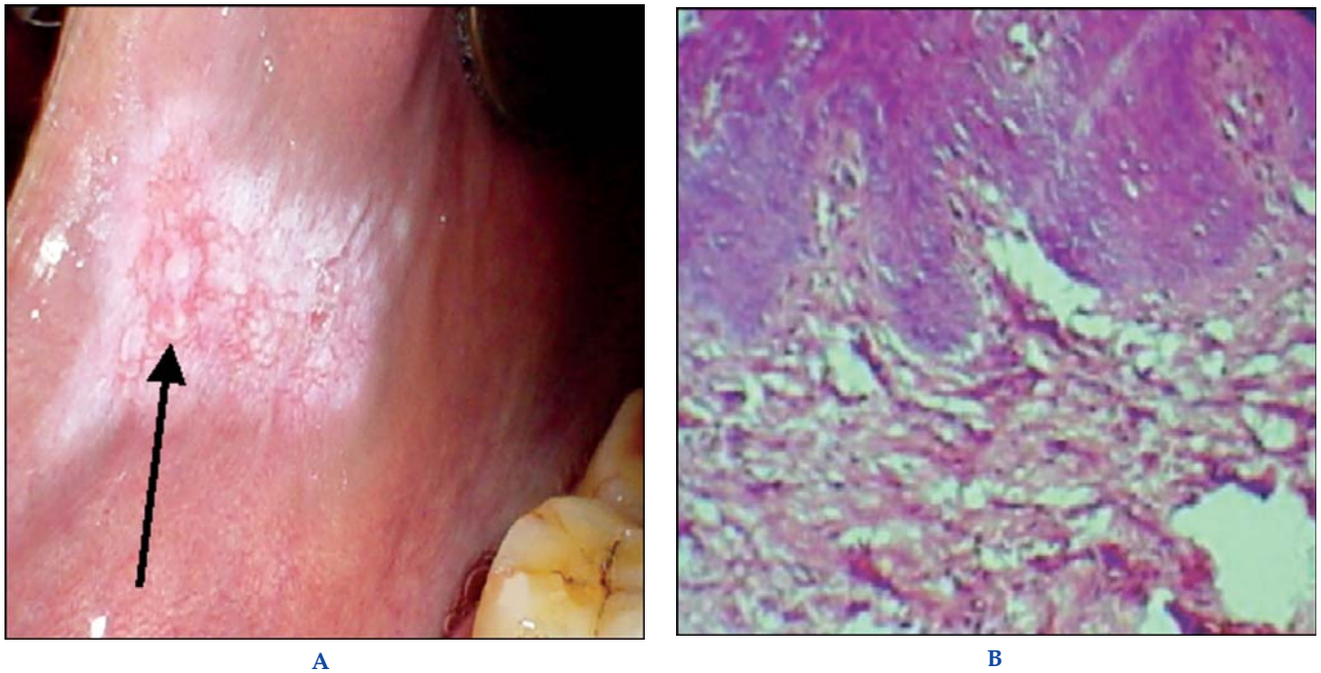
Pre-Leukoplakia (Mehta et al (1971):[10] It has been described as a low grade or mild reaction of the mucosa occurring as gray or grayish white but never completely white lesion with a slight globular pattern and indistinct borders blending into adjacent normal mucosa (Fig. 12.4).

Homogenous, nodular or speckled, Combination, Erythro-Leukoplakia, Verrucous Leukoplakia, Hairy Leukoplakia are the different clinical variants.[18]

Homogenous: White plaques have no red component but have a fine white grainy texture or more mottled rough appearance (Figs 12.5 and 12.6).

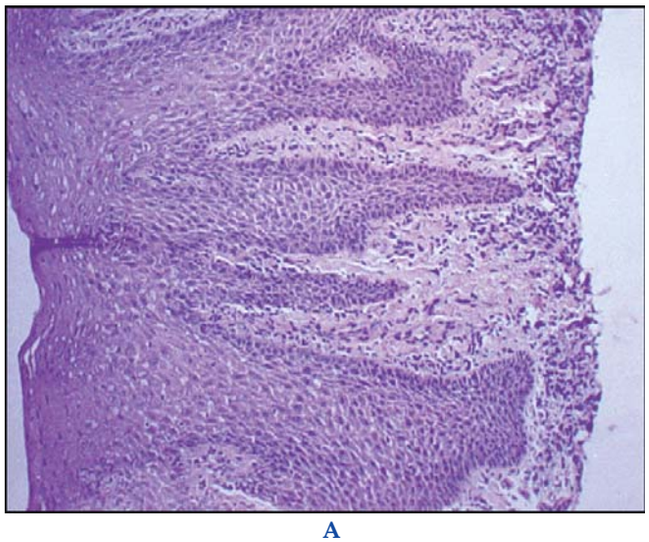
A B

FIGURES 12.6A and B: Showing diffuse area of whitish discoloration on the buccal mucosa. The lesion was unscrapable. This patient 22-year-old male with habit of smoking *bidi* and cigaratte since 8 years. Diagnosed as homogenous leukoplakia. (Omal PM, Beena K, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)



A B

FIGURES 12.7A and B: Showing (A) Nodular leukoplakia on the angle of the mouth. It appears as a mixed red and white lesion, predominantly white with small nodules scattered on the lesion. It is of special importance as it has high risk of changing into malignancy. (B) Histological photograph showing dysplastic features to be added (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)



A

B

FIGURES 12.8A and B: showing left side the Histopathological slide of the 32-year-old female with only arecanut chewing habit of 8 years. Patient was severely anemic. The red white lesion was extremely painful and clinically diagnosed as Speckled Leukoplakia (Ani John, Umarji H GDC Mumbai 2004)

Speckled Leukoplakia: Composed of white and red flecks of either fine or coarse variety. Combination of red and white patches, may be termed as the ***Erythroleukoplakic*** lesions (Figs 12.7 and 12.8).

Verrucous leukoplakia: Possesses red and white component also but the white component is much thicker and thrown into folds and protrudes above the surface mucosa.

All the clinical types have the ability to change from one type to another and so considering them as totally discrete entities would be erroneous.

Hairy Leukoplakia is seen on the lateral aspect of the tongue in sero-positive cases of AIDS, this lesion has white corrugated sharp tapering projections; the appearance may be changed by local trauma and infection.

Reversible and Irreversible is another way of classifying Leukoplakias, depending on whether or not they subside on cessation of habits, and therapeutic intervention by the clinician.

The term cryptogenic leukoplakias/Idiopathic is used when no underlying risk factors are identified. In Smith et al study[6] 1.3 percent out of 6700 cases were of this variety. They have been reported to have higher malignant potential.[6,7] Hungarian study, however, reveals a much higher 4.2 percent out of 520 cases, of idiopathic leukoplakia.[9]

Leukoplakia associated with ulcers: Schepman K et al[29] 1999 found in their series that almost 50 percent of oral squamous cell carcinomas are presumably associated with or preceded by leukoplakia. They opined that early detection and active management of patients with oral leukoplakia might prevent the true development of a number of OSCC.

Histologic Types

- Those that show no atypia or dysplasia
- Those that show differing degree of atypia

List of Histopathologic Features that we look for in the Light Microscopy of Leukoplakia

- Hyper ortho keratosis/Hyper para Keratosis
- Acanthosis
- Dysplastic criteria
- Abnormal mitosis
- Individual cell keratinization
- Epithelial pearls within the spinous layer
- Alteration in the nuclear cytoplasmic ratio

- Loss of polarity and disorientation of cells
- Hyperchromatism of cells
- Large prominent nucleoli
- Diskaryosis or nuclear atypism.. Giant nuclei
- Poikilocarynosis—division of nuclei without division of cytoplasm
- Basilar hyperplasia Carcinoma-in situ or Intrapithelal carcinoma.

Classification and Staging

Clinical and histopathological features of leukoplakia are suggestive of pre-malignant potential. Early detection and management is a continuing goal. Recently classification of leukoplakia considering both the features is being advocated as it promotes uniform reporting and management strategy. Given below is the modified LCP classification given by van der Waal et al.[33]

L1 Size of single/multiple leukoplakia together ≤ 2 cm
L2 Size of single/multiple leukoplakia together 2 to 4 cm
L3 Size of single/multiple leukoplakia together ≥ 4 cm
LX Not specified
PO No epithelial dysplasia
P1 Distinct epithelial dysplasia
PX Not specified

STAGING	
STAGE I	–L1 P0
STAGE II	–L2 P0
STAGE III	–L3 P0 OR L1/L2 P1
STAGE IV	–L3 P1
NOTE: IF P *IS NOT AVAILABLE* C *(CLINICAL TYPE) IS TAKEN FOR STAGING*	
STAGE I	–L1 C1
STAGE II	–L2 C1
STAGE III	– L3 C1 OR L1/L2 C21
STAGE IV	– L3 C2
C1 = *HOMOGENOUS* ; C2 = *NONHOMOGENOUS*	

Treatment of Leukoplakia

Once leukoplakia is recognized by the dental surgeon, he should classify it into the non speckled and speckled.

Check local factors of irritation.

1. Sharp teeth, prosthesis cheek bites, correct them.
2. Habits of tobacco and alcohol abuse to be counseled for stopping or atleast marked reduction.
3. Systemic factors of vitamin deficiency, candidiasis, anemia, or syphilis should be screened by specific tests and corrective measures taken.

van der Waal I et al (1997)[31] have said it is preferable to use the term leukoplakia as a clinical term only. When a biopsy is taken and report obtained histopathologic description should replace this clinical label. The degree of dysplasia will determine how much dangerous the lesion would be.

Transformation of oral leukoplakias into malignancies overall rate of transformation.1% to 9% average 3%

Floor of the mouth	.43%
Tongue	24%
Lips	24%
Palate	19%
Buccal Mucosa	17%
Retromolar area	.12%

Data from Waldron CA; Shafer WG: Clinico-pathologic study of 3256 oral leukoplakias, Cancer, 36: 1386, 1975.

1. Surgical stripping in stages with free grafts or else with allowance for the denuded surface to epitelialize by secondary healing.
2. Large or widely disseminated lesions when excised leave large surgical wound, skin grafts may be used.
3. Cryosurgical procedures have been used for large lesions with good results.
4. Laser surgery has been used recently, long-term effects of such a surgery is yet unestablished. Schoelch ML et al (1999)[30] treated seventy leukoplakia lesions with CO2 and Nd:YAG lasers, and standard laser safety protocols were used. There was no postoperative infection, hemorrhage, or paresthesia. Two patients developed pyogenic granulomas in their surgical sites. Verrucous lesions had an especially high rate of

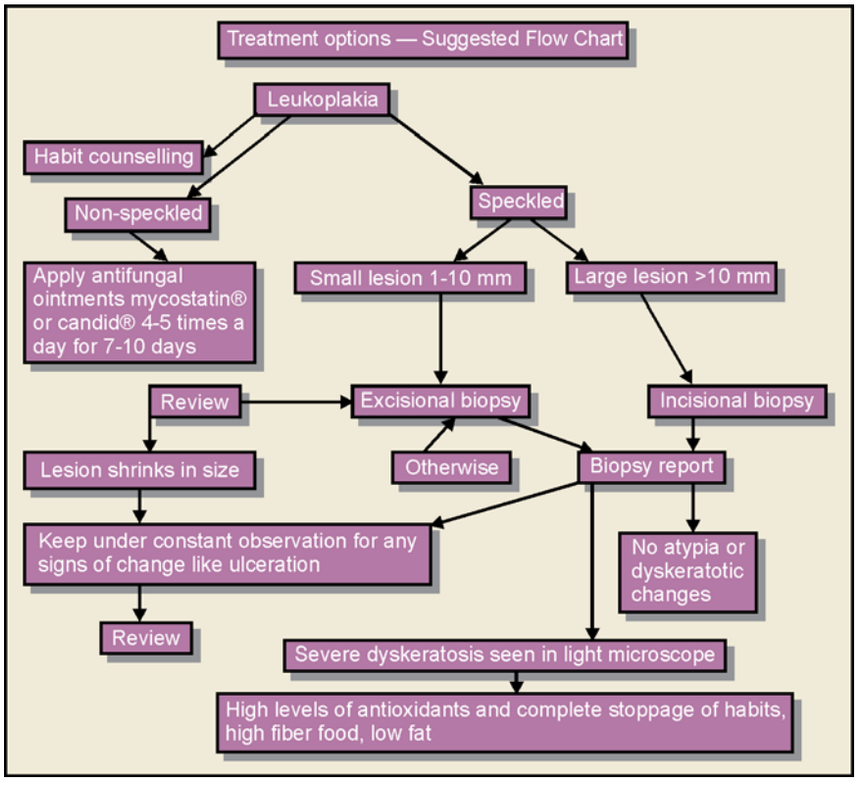


FIGURE 12.9: High levels of antioxidants and complete stoppage of habits, high fiber food, low fat

recurrence (83%). Laser surgery of oral leukoplakia according to them is an effective tool in a complete treatment of most leukoplakias.

5. Antimetabolities cream like 5FU (5-Fluorouracil) has been used with moderate success, long term follows up studies are yet to come in.
6. Artificial analogs of vitamin A called as retinoids—(13 cis retenoic acid, tretinoin, etc.) to be applied locally for two to three weeks. At present there is no evidence to justify the systemic use of Vitamin A in the treatment of leukoplakia.

Epstein JB and Gorsky M (1999)[32] evaluated the use of topical 0.05 percent vitamin A (tretinoin) acid gel for the treatment of oral leukoplakia. Tretinoin was applied topically 4 times a day for the management of nonmalignant oral white lesions in 26 patients. The use of topical vitamin A acid showed a limited effect in controlling oral leukoplakia. Further studies are needed to establish the appropriate indication.

7. Symptomatic treatment—LA cream (Fig. 12.9)

Place	*Year*	*Author*	*Patients*	*% of malignant Transformation*
Ernakulam	1980[11]	Gupta et al	410	2.2%
Bhavnagar	1980[11]	Gupta et al	360	0.3%
Bombay	1971[12]	Gangadharan and Paymaster	626	10.0%

Lichen Planus

The name lichen planus refers to the superficial similarity of the lesions of the reticular lichen planus to a lace like pattern produced by symbiotic alga and fungal colonies on the surface of rocks in nature, termed as lichens.

Etiology of Lichen planus is not yet fully elucidated and the following theories must be considered.

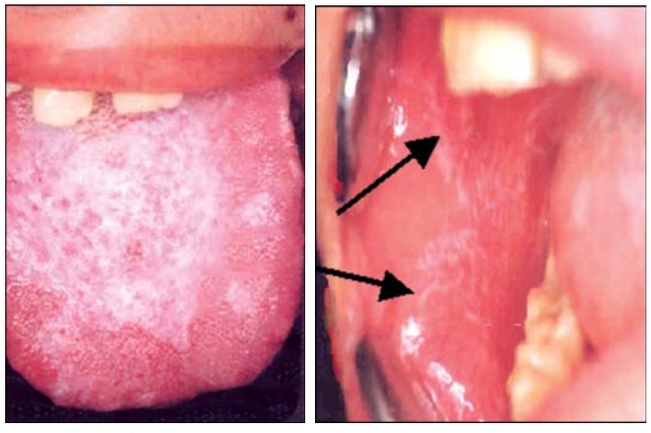
I. Immunologically induced degeneration of basal cell layer of epithelium is the prime suspect.
II. It is postulated to have a strong psychosomatic background in its etiology.

III. Lesions are associated with chronic drug ingestion and are termed as lichenoid reactions, however, they are not clinically distinguishable from lichen planus.
IV. It is possible to inter-relate the 1st and the 2nd point since strong psychosomatic stresses can result in autoimmunity reactions according to many of the recent studies in psychoneuro-immunology.

Clinical Features and Types (Reticular Type)

It appears as a striated, linear papular lesion whose edges mostly have fine striae called as the *Wickhams striae.* They form lace like interlacing or annular forms. Usually bilaterally symmetrical, involving cheek and tongue commonly, but can occur anywhere (Fig. 12.10). It is a disease of adulthood and slightly more prevalent in the women. Burning sensation varies from none to severe. More than 50 percent are showing some associated skin lesion. Skin lesions are occurring on flexor surfaces of wrists and forearms and inner aspects of knees and thighs, in the trunk the sacral area (Fig. 12.11). Face may remain uninvolved in the most of the cases. Primary symptom is itching on the skin. Lesions are small angular flat-topped papules, covered by glistening scales, few mm in diameter and gradually coalesce into larger plaques. Banoczy[13] has mentioned different clinical types of oral lichen planus.

FIGURE 12.10: Figure showing annular type of Lichen Planus of the buccal mucosa in a 65-year-old male patient who used to stay in a old age home. However, he did not have any systemic lesions but he complained of burning sensation in the mouth. This person was treated with minor anxiolytics (Nillofer S, Prasanna K, Bailoor DN, 2003, Yenepoya Dental College Hospital, Mangalore)



A **B**

C

FIGURES 12.11A to C: Showing varied clinical appearance of Lichen Planus. (A) Characteristic interlacing, slightly elevated, fine whitish lines (Wickhams striae), lace like lesion on the tongue. (B) Wickhams striae, reticular type on the buccal mucosa of the same patient. This was a 47-year-old female patient who had severe pruritic lesions on the skin, i.e. on the hands and legs as shown in (C) patient was later treated with systemic and local corticosteroids with consultation of a dermatologist (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College Hospital, Mangalore)

Reticular type	51.5%
Erosive type	27.6%
Atrophic and Papular type	12.6%
Bullous type	8.3%

Atrophic type. Inflamed areas of oral mucosa with edges of lacy lesions usually tend to ulcerate and form the erosive type. In India we usually see the atrophic and erosive occurring concomitantly and are associated with symptoms like pain and burning. Cheeks and tongue, and the vestibular regions appear to predominate in our series of patients. Bullous forms are seen very rarely in the Indian populations and once it ruptures it may resemble or transform into the erosive type. Hypertrophic forms have been mentioned and these have lesions thrown into folds and only histopathologic examination can reveal the true nature of the lesion.

Histopathology

- Hyperparakeratosis
- Hyperorthokeratosis, with thickening of granular layer
- Acanthosis with intracellular edema of the spinous cells
- Saw tooth appearance of rete pegs
- Liquefaction degeneration of basal layer
- Civatte bodies (Colloid bodies present in epithelium).

Association of Lichen planus, with Diabetes mellitus and Hypertension was described as the *Grinspan syndrome*. Recent studies have not confirmed the associations, and in India also clinically these associations have not been noted. It is possible that the drugs taken, long term, for hypertension and diabetes could probably give a lichenoid reaction leading to the belief in such a triad.

In our department we clinically stage the Lichen planus into three groups.

Group 1: Lichen planus of reticular, atrophic variety. No symptoms discovered on routine oral examination.

Group 2: Lichen planus of reticular, atrophic or any other variety except the erosive (a) With mild to moderate pain/burning (b) With moderate to severe pain/burning.

Group 3: Erosive lichen planus with or without symptoms.

Group 4: Patients who are on regular drugs continuously for more than six months may show signs of lichenoid reaction. (Burning sensation mild-severe).

Treatment

Psychological testing with simple questionnaire type formats is recommended in patients in all groups. Hospital Anxiety and Depression Inventory is the most commonly used. There are a high percentage of anxiety prone and tense individuals having these lesions.

Group 1: Here usually no treatment is the best treatment. Regular follow up and with 2mg/5mg Diazepam for anxiety alleviation is recommended. Patient should be informed about the nature of the disease. Dhruva and Ani J[26] mention application of topical vitamin A as effective for management of this stage lesion.

Group 2: Requires local application of gels like Benzocaine 10% (Mucopain®) to relieve burning or Cream Xylocaine (Lignox®). Counter irritant salicylate gels must be avoided. 2 mg diazepam for two to three weeks is recommended for this type for anxiety relief. Ointments, salves containing corticosteroid applied locally 3 to 4 times a day but considering the effect of the saliva and its washing out, its actual local effect could be questioned.

Injection of steroids intra lesionally (Three times a week for six weeks is normally recommended.) for quicker relief, by multiple puncture method in the submucosal area. In case of no training in this method it is best left to a specialist of this field. 4mg/ml vial of Betnesol® to be used for one lesion. Systemic steroids are not warranted considering the risk versus the benefit. Regular follow up is necessary.

Group 3:

- Immediate biopsy of the lesion is indicated.
- Local control of pain with Benzocaine or Xylocaine.
- If the ulcer appears infected then a Penicillin group of antibiotics like Pentids®, or OracynK® could be prescribed for 4 days.
- If biopsy report shows any evidence of premalignancy then immediate referral to a Oncology unit is mandatory.

Group 4: Discontinuing the drug with physicians consent, and local application of the Benzocaine 10 percent cream would normally give results. Patch testing to the local dental restorative materials is advised.

Differential Diagnosis

It must be differentiated from the following lesions in the oral cavity.

- Leukoplakia
- Electrogalvanic mucosal lesions
- Linea alba bucalis
- Leukoedema
- Ectopic geographic tongue
- Lupus erythromatosus
- White sponge nevus.

Machado AC et al[34] evaluated the efficacy of topical, systemic and/or intralesional corticosteroids in the management of symptomatic cases. Fifty-two patients with OLP, 33 females and 19 males, aged from 17 to 75 years. Symptomatic lesions were seen in 29 patients were treated with corticosteroids. Asymptomatic cases were only clinically monitored. They found topical corticosteroid therapy alone was effective in producing relief of symptoms. For lesions non-responsive to topical treatment, they have advocated the use of intralesional injection and/or short-term use of systemic corticosteroids.

Erythroplakia

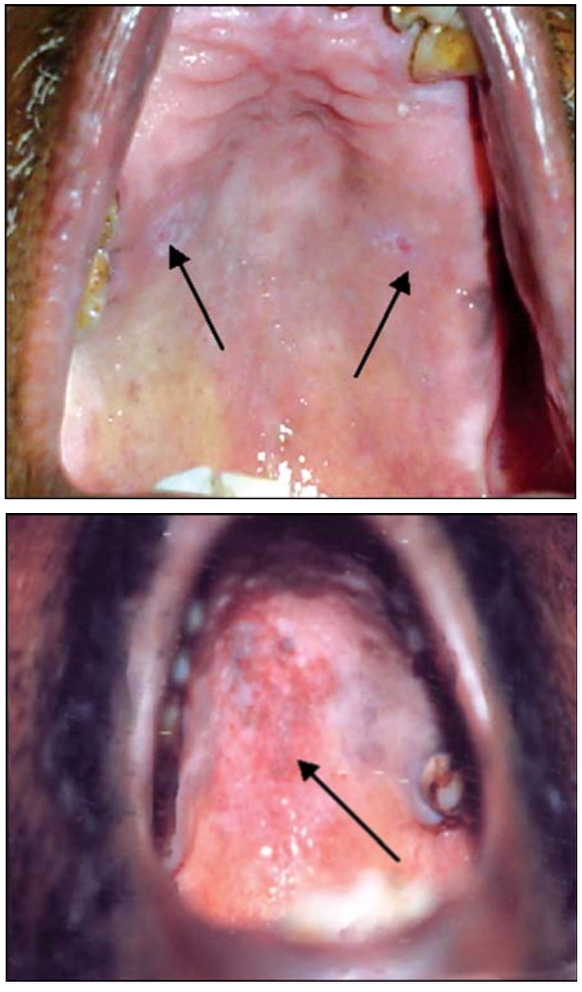
Term applied to any area of reddened, velvety textured mucosa that cannot be identified on the basis of clinical and histopathologic examination as being caused by inflammation or any other disease process.

Clinical Variants

Homogenous erythroplakia interspersed with patches of Leukoplakia and granular or speckled erythroplakia. All clinical variants have a high rate of premalignancy and is not solely a feature of speckled erythroplakia, 80 to 90% of erythroplakias are histopathologically either severe epithelial dysplasia, carcinoma *in situ* or invasive carcinoma. In view of the clinical significance, the differential diagnosis is critical.

- Candidiasis
- Lupus vulgaris
- Histoplasmosis
- Areas of mechanical irritation.

It has no sex predilection, usually seen in 6th or 7th decade. Risk factors are unknown, although strong correlation with alcohol and tobacco abuse is seen. Prime areas of erythroplakia development are floor of mouth, ventral surface of tongue, soft palate, anterior faucial pillars. To differentiate the erythroplakia with malignant change and benign inflammatory lesions, 1% toludine blue or toludine chloride is applied with swab or oral rinse. Drying of oral mucosa before the examination for Ca screening is important. One percent acetic acid rinse after application of toludine blue solution usually eliminates non specific reactions which occur from mechanical retention of stain. Erythroplakic lesions retain the stain. This gives quite good results. The floor of the mouth



A

B

FIGURES 12.12A and B: Showing (A) whitish nonscrapable areas on the palate in a habitual *bidi* smoker. Indicative of leukoplakia. (B) Reddish white discoloration associated with burning in a 53-year-old male who had habitual pan chewing and cigarette smoking since 35 years indicative of erythroplakia (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College Hospital, Mangalore)

appears to be the most common site affected in males while mandibular gingival and alveolar mucosa, mandibular sulcus is common in females. Principles of treatment is same as leukoplakia. Prompt biopsy appears to be the correct clinical procedure with malignant lesions referred to the oncology department without much delay (Fig. 12.12).

Stomatitis Nicotina

Specific type of leukoplakia, seen in *bidi* and pipe smokers, in fact in all heavy smokers. Parboiled appearance, because of many transacting wrinkles and fissures which divide the white mucosal surface into small nodular areas. Red dot is situated in the middle of each nodule and represents the inflamed orifice of a minor salivary gland duct. Its one of the reversible leukoplakias and has low malignancy conversion (Fig. 12.12).

Differential Diagnosis (Buchner and Sandbank)[15]

Papillary hyperplasia, Darriers disease, focal epithelial hyperplasia, Goltz's syndrome, Cowden syndrome, acanthosis nigricans, multiple neuroma syndrome, multiple oral fibromas in tuberous sclerosis, multiple papilloma, multiple condylomas.

Snuff Dippers Lesion

It is a pre-leukoplakic lesion seen commonly in the right buccal vestibule, lower labial vestibule, seen in relation to area of placement of quid in persons with chewing habits.

Electrogalvanic White Lesions

Presents as leukoplakia, lichenoid or oral discoid lupus erythematosus lesions. If lesions come after inserting a metallic restoration then this diagnosis must be considered and if more than a decade elapses then it is difficult to reverse this lesion, Banoczy's[14] and Lundstrom's[16] study says that the corrosion of the amalgam and the gold filings were having a high correlation with erosive type of lichen planus.

White Hairy Tongue

Caused by elongation of the filiform papillae because of increased retention of keratin. Dorsal surface of the tongue affected, mostly in males, chief complaint of gagging and halitosis may be present, colors may vary with tobacco, food consumption, smoking and candidiasis super infection. Farman[17] is of opinion that patients with malignant neoplasia are more prone to develop this condition.

Treatment

Careful frequent brushing with soft tooth brush or in severe cases clipping of the elongated papilla. Topical keratolytic agents and yogurt or lactobacillus acidophilus cultures have been reported by Brightman[18] to give relief. Use of systemic antibiotics and topical medications, hydrogen peroxide perborate and similar oxidizing agents may cause this lesion.

Papilloma

A benign tumor of epithelium. Human papilloma virus has been implicated, Loning et al[19]. It's a exophytic lesion with characteristic papillomatous shape. Lesion is always pedunculated and has a rough cauliflower like, pebbly surface. It is either pink or white in color. Greer and Goldman[20] reviewed 110 cases and found tongue (33%) and in descending order of occurrence, palate, buccal mucosa, gingiva, lips, etc. Age 21-50 average 38 years. Histopathology is characteristic.

Differential Diagnosis

Verruca vulgaris, Papillary sq cell carcinoma, verrucous ca, condyloma acuminatum, Condyloma lata, Pseudoepitheliomatus hyperplasia.

Management

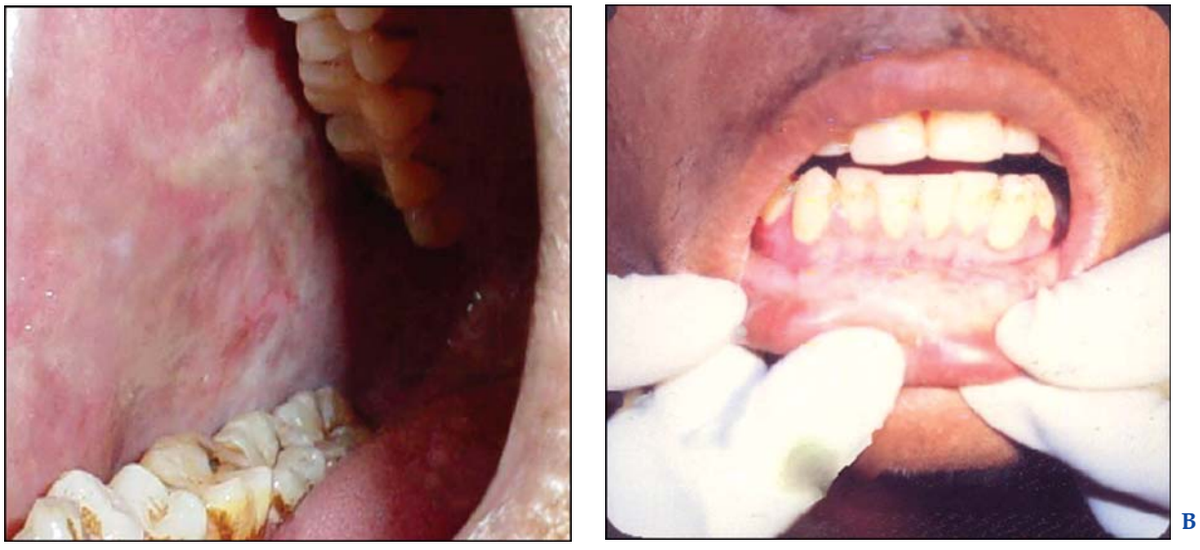
Surgical excision seems to be the best treatment.

White Sponge Nevus

It is a hereditary condition in which white lesions occur on various mucous lesions of the body. It has autosomnal dominant inheritance pattern. Present at birth, intense at puberty.

Features

Variable-mild just occurring on the buccal mucosa to severe covering all the possible areas it is asymptomatic and has



FIGURES 12.13A and B: Showing (A) Blanching of buccal mucosa with central area of erosiveness. (B) Labial mucosa appears white and stretched. Case of oral submucous fibrosis grade III. This patient was a 20-year old college going student who had taken to gutka chewing due to peer pressure (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College Hospital, Mangalore)

no premalignant-potential. Diagnosis is established by correct history taking.

Differential Diagnosis

Leukoedema, leukoplakia, lichen planus, pachyonychia congenital hereditary benign intra epithelial dyskeratosis (HBID). Proper identification and allaying the patients concern is the best treatment.

Skin Graft

In Caucasians the skin grafts appear to be confused with white lesions by the uninitiated doctors but in India such a confusion is unlikely since the pigmentation in the skin as well as the patients surgical history will reveal the obvious.

Oral Submucous Fibrosis

This is a slowly progressive disease in which fibrous bands form in the oral mucosal leading to severe restriction of movement of the mouth including the tongue, seen in the people of the Indian subcontinent and also in the Indian populations in Africa, Fiji, UK etc.[2] Burning sensation of variable intensity felt. Entire oral mucosa is blanched and pale in appearance, occasionally it presents with vesiculation of the mucosa. The etiology is yet to be established (See Chapter-Oral Precancer for details) (Fig. 12.13).

Verrucous Carcinoma

It is a type of slow growing low-grade carcinoma of the oral cavity. Clinically it looks like a cauliflower, or similar rough pebbly surface thrown into folds. Average age of male=64.7 females=71.0. Slight predilection for females noted. Buccal mucosa, alveolar ridge, gingival, tongue is other sites involved. Histopathology is characteristic with broad blunt rete ridges, which demonstrate pushing borders. It is frequently associated with leukoplakia, epithelial dysplasia and with squamous cell carcinoma.

Candidiasis

Is an infection by dimorphic yeast like fungus *Candida albicans*. This fungus exists in a commensal relationship normally in the oral cavity. This is a low virulence organism, which has interdependent metabolism with adjacent flora.

For overgrowth of Candida-

- Competitive flora should be reduced for example use of topical antibiotics.

FIGURES 12.14A and B: Showing 58-year-old male patient with renal disease and on immunomodulatory drugs who had candidal lesion on the tongue (Prasanna K, Nillofer S, Bailoor DN 2004, Yenepoya Dental College Hospital, Mangalore)

FIGURES 12.15A to C: Showing 48-year-old male patient who was a chronic smoker with scrapable diffuse white lesion on the dorsum of the tongue. Smear preparation shows candidal hyphae (Nillofer S, Prasanna K, Bailoor DN 2004, Yenepoya Dental College Hospital, Mangalore)

- Drastic reduction in the resistance of the tissues. This reduction could be generalized as in AIDS or localized as in angular cheilosis. Refractory candidiasis normally indicates underlying debilitating disease.

Dreizen et al (1983)[24] reports 70% of infection in patients undergoing chemotherapy were caused by *C. albicans.* Prolonged steroid therapy too, is associated with severe candidal infection.

Moskow et al (1972)[25]

- *Basic types of clinical lesions-*
- Pseudomembranous white lesions
- Chronic hyperplastic white lesions
- Atrophic red lesion.

Clinical Features

Symptoms vary from painless to burning sensation to severe tenderness. Pseudomembranous infection shows as fine whitish deposits on the oral mucosa, which leaves raw bleeding surface on wiping. Often the entire mucosa appears diffused red as food mastication removes this curd like lesions (Fig. 12.14).

Treatment

- Ointment Mycostatin—to apply 5 times a day for 10 days.
- Solution Fungistat—to apply locally
- In systemic need—Tablet Fungazole-Tablet Ketoconazole 200 mg
- Mycostatin tablets—-500,000 units of Nystatin 1 tablet 3 times a day orally.

Chronic Hyperplastic Candidiasis

Keratotic lesion, this cannot be scraped off. In case of low-grade chronic infections by *Candida albicans*, the yeast products may not be sufficiently concentrated to coagulate the surface epithelium but rather may stimulate the production or retention of keratin. This lesion then would resemble more a leukoplakia than Candida (Fig. 12.15). Holstrup, Bessermann[21] have mentioned the association with chronic multifocal candidiasis. Cawson and Binnie[22] have shown in their series a definite relationship between chronic candidiasis and oral epidermoid carcinoma and postulated the former to be the causative factor for the latter. Refractory angular cheilitis appears to be associated with this lesion and Bjorlin[23] mentions the effectiveness of surgical treatment in such cheilitis. Local application by Nystatin cream usually results in the regression of the lesion.

Squamous Cell Carcinoma

This also appears as a white leukoplakia like lesion but it invariably contains a large ulcerated component with indurated edges. Oral cancer is dealt in detail in a separate section.

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13 Vesiculo-bullous and Ulcerative Lesions of Oral Mucosa

INTRODUCTION

The practicing dentists commonly see this group of lesions and the diagnosis has a frightening range from an innocuous traumatic ulcer, which magically resolves to the malignant carcinomatous ulcer, which kills. Various listings and classifications help us to focus our thoughts on the differential diagnosis and the mental ticking off process to arrive at the basic group of the lesion. Then subsequently we narrow it down to one or two of that on basis of laboratory investigation.

- **VESICLE** is an elevated blister containing clear fluid that is under 1cm in diameter.
- **BULLAE** are elevated blister like lesions containing clear fluid that are more than 1cm in diameter.
- **ULCER** is defined as a complete discontinuity of epithelium caused by pathological processes in distinction to the Erosion which is a partial destruction of epithelial structures with intact basal cell layers.

CLASSIFICATION

1. *Classification I*
   - I. *Hereditary*
     - a. Epidermolysis bullosa EB
   - II. *Traumatic*
     - a. Traumatic ulcer
   - III. *Allergic*
     - a. Stomatitis medicamentosa
     - b. Stomatitis Venenata
     - c. Angioneurotic edema
   - IV. *Viral*
     - a. Herpes simplex IRHS
     - b. Herpangina HRP
     - c. Hand-Foot-and-Mouth (HFM) disease
     - d. Hoof and mouth disease HM
   - V. *Autoimmune deficiency*
     - a. Erythema Multiform EM
     - b. Acute Epidermal Necrolysis AEN
     - c. Stevens Johnson Syndrome (SJ Syndrome)
     - d. RAU-Major-Minor-Herpetiform
     - e. Behcet's disease
     - f. Pemphigus vulgaris
     - g. Pemphigus vegetans
     - h. Bullous Pemphigoid
     - i. Cicatrical Pemphigoid
   - VI. *Neoplastic*
     - a. Oral Sqamous cell carcinoma
     - b. Ulcers related to bone marrow depression (Refer Chapter 17 for detailed description)
   - VII. *Miscellaneous*
     - a. Cyclic Neutropenia ulcers
     - b. Erosive Lichen Planus

2. *Classification* II
   I. *Non-recurrent*
      a. Single—traumatic
      b. Multiple—Acute herpetic
   II. *Recurrent*
      a. Single—RAU
      b. Multiple—Chronic ulcerative stomatitis

EPIDERMOLYSIS BULLOSA (EB)

It is a rare hereditary disease in which bullae form on the skin and mucous membranes following mild to moderate trauma.

Areas subject to frequent mild trauma such as the elbow, and knees develop bullae which subsequently rupture.

In the milder type of the disease, (epidermolysis bullosa simplex) healing takes place without scaring. In the more severe type, epidermolysis bullosa dystrophica mutilating atrophic scars are produced.

Epidermolysis bullosa simplex improves at about puberty, whereas EB Dystrophica is a progressive lesion and involves the mouth and pharynx as well. Dental treatment of these patients is very difficulty since inadvertent pressure on the lips and oral tissues causes a bullous lesion or ulcer to form. In very severe cases, death occurs in the first week. Definitive treatment is not known. Symptomatic relief may be given by use of Local anesthetics, and Steroids give relief in some cases.

TRAUMATIC ULCER

Most common oral ulcer-cause may be mechanical, chemical or thermal, accidental, self-inflicted or treatment related.

Features: Pain is a common presenting symptom—the ulcer of variable size appears in the mucosa adjacent to the irritating factor like clasp of a partial, orthodontic appliance, ill fitting complete denture, tooth brush injury-due to careless and repeated brushing with hard brush. Mader (1981)[5] discussed the occurrence of a lingual frenum ulcer caused by oro-genital sex. Their borders are somewhat raised and reddish, and their bases have a yellowish necrotic surface that can be readily removed.

Traumatic ulcers on vermillion border have a crusted appearance. Secondary infection may confuse the picture and give rise to cervical tender lymphadenopathy.

Budtz-Jorgenson (1981)[6] mentioned that the denture induced traumatic ulcers were observed in 5.5 percent of a population in the sixth and seventh decade of life. Most of the traumatic ulcers become painless in 3 to 4 days after the injury and most heal within 10 days, unless secondarily infected. Orabase with Kenalog® is given as a choice of treatment in most books. It is better to advice the patient about using Hexigel® Ointment, Hexidine mouth wash or even Mucopain® ointment to relieve the acute symptoms of pain. The primary cause, however must always be located and removed.

Only in immunocompromised cases severe secondary infection may make the need of antibiotics mandatory.

A persistent ulcer not responding to the foregoing regimen should be surgically excised and will heal with primary closure and the entire tissue must be sent for histopathologic examination to rule out dysplastic changes.

ALLERGIC REACTIONS

An allergic reaction in the oral cavity is most often seen to drugs. It may be erythematous, vesicular or ulcerative. An uncommon type of drug reaction is angioedema.

Angioedema

It is an aquired or hereditary, soft tissue deffuse painless swelling usually involving lip, neck or face. Drugs or food items may precipitate the allergic reaction. In some cases it may require emergency treatment because of respiratory distress. Antihistamines and corticosteroids are used if allergy is a causative factor (Figs 13.1 and 13.2).

VIRAL

Primary Herpetic Gingivostomatitis (Intraoral recurrent herpetic stomatitis)—IRHS.

There are two main types of herpetic infection—primary and recurrent. This infection caused by herpesvirus hominis is common cause of multiple ulcerations. Most of the Indians (at least 75%) have antibodies to type I herpes

FIGURES 13.1A and B: Showing 27-year-old female who took an OCT drug without prescription for tooth ache and presented to the dentist with a painless swelling of the lower lip. This is presented as an atypical presentation of angionuerotic edema of the lower lip. As many as 45% of the patients in India take medications directly from the pharmacies without prescription due to lax implementation of existing laws. She responded well to Tab Betnesol® 0.5 mg twice a day for three days with immediate discontinuation of the offending drug and dental treatment to alleviate the pain (Bailoor DN, Prasanna K, Nillofer S 2003, Yenepoya Dental College and Hospital, Mangalore)

FIGURES 13.2A and B: Showing pre and post picture of young boy with Angioedema (Bailoor DN, Prasanna K, Nillofer S 2004, Yenepoya Dental College and Hospital, Mangalore)

simplex virus. In primary, vesicles appear on Vermillion border, with acute onset of fever, headache, irritability, and painful lymphadenopathy. Vesicles are also seen in cervical region. These vesicles rupture, becoming pinpoint ulcers and lip becomes bloody and crusted and in children the saliva may drool. Approximately 33 percent of these patients subsequently develop recurrent herpes labialis. Recurrent herpetic stomatitis develops in adolescents or elderly on reactivation of the dormant virus.

The dental surgeon should be aware of the possibility of getting herpetic whitlow on his fingers. Merchant et al[2] 1983 mention the use of the disposable gloves, rubber dam, and autoclaving it prior to throwing in the trash. Use of safety glasses is generally recommended to guard against herepetic infections of the eyes.

Diagnosis: The diagnosis of PHG is made on clinical basis, since clusters of ulcers are pathognomonic in a bed of inflamed mucosa. Virus may be isolated in tissue culture provided that intact vescicle was freshly punctured. But in most Indian villages studying antibody titer and tissue culture is a far cry from reality.

Investigation:

Cytology: A fresh vesicle can be scrapped from the base of the lesion and placed on a microscopic slide. The slide is stained with giemsa and searched for multi nucleated giant cells, synctium and ballooning degeneration of the nucleus.

HSV Isolation: Isolation and neutralization of a virus in tissue culture is most positive method of identification. Rabbit kidney and human amnion are both sensitive to HSV. Isolation of HSV from oral lesions does not necessarily mean that HSV caused the lesions. Patients who have lesions from other causes may also be carriers of HSV antibody.

Antibody titers: An acute serum specimen should be attained within 3 to 4 days of the onset of symptoms. The absence of detectable antibodies plus the isolation of HSV from lesions is compatible with the presence of a HSV infection. Antibody to HSV will begin to appear in a week and reach a peak in 3 weeks.

Differential diagnosis: Herpangina-Coxsackie A virus, affects children, late summer and early monsoon season, in soft palate and faucial area, fever malaise.

Hand foot and mouth disease— also caused by Coxsackie A virus children under 10 year of age-nausea-diarrhea-fever-vesiculo-ulcerative lesions occur simultaneously in oral cavity, and on the hands and feet.

Management: Both the RAU and the IRHS heal in 8 to 14 without any Rx, however following regimen is used in most centers in India.

- Symptomatic ?
- Specific

Symptomatic: Topical anaesthetic like

- Lignocaine Viscus ®
- Mucopain® ointment
- Dentogelointment®

Topical antiinfective to prevent secondary infection

- Hexigel®
- Mouth wash Hexidine®
- Tetracyline mouth wash 4 times a day for 4 days.
- Elixir of Diphenhydramine (antihistaminic).

Specific:

- Zovirax® Acyclovir is effective as cream in immunocompromised patients.
- Syrup-Panadol® Paracetomol, Crocin® etc. to control fever.
- Proper Oral hydration using tender coconut water which is easily available and effective rural remedy for fever rehydration. In children with severe hyperpyrexia intravenous liquids with B complex serves to put the patient back on his feet.
- Infected ulcers appear to benefit from the tetracycline mouth rinses and subsequent application of kenalog in Orabase® which is the only adhesive paste available in India.
- High doses of Lysine have been known to act prophylactically according to studies by Thein and Hurt[4] and reduce the time of healing and suppress the recurrences.

Antibiotics are of no help and corticosteroids are contraindicated. Anti viral agents have been utilized with

success. Idoxiuridine (IDU) Cytosien arabinoside (Ara-C) and adenine arabinoside (Ara-A) have been used systemically. In view of the severe hepatic and renal toxicity of these agents and the fact that they are very expensive today, it seems that these should only be used in special cases where HSV infection is associated with immune deficiency.

FIGURE 13.3: Showing viral wart in a patient who developed it after exposure to sun on the beach.(Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

Herpes Zoster

Etiology

Varicella zoster is a DNA virus similar to HSV and causes both a primary and recurrent infection and remains latent in the nerve tissue. Chicken pox is a generalized primary infection that occurs the first time an individual contacts the infection. After the primary disease is healed, the varicella zoster virus becomes latent in the dorsal root ganglia of spinal nerves or extra medullary ganglia of cranial nerves. The varicella zoster viruses become reactivated in some individuals causing lesions, of localized herpes zoster.

Clinical Manifestations

General Findings: Chicken pox is a childhood disease characterized by mild systemic symptoms and a generalized intensely pruritic eruption of maculopopular lesions that rapidly develop into vesicles on an erythematous base.

The lesions may be confined to the mouth and face. A prodromal period of 2 to 4 days with shooting pain, paraesthesia, burning and tenderness appear along the courses of the nerve. Unilateral vesicles then appear in clusters chiefly along the course of the nerve giving the characteristic clinical picture of single dermatome nerve

FIGURES 13.4A to C: Showing 18-year-old boy developed acute unilateral vesicles intra and extraorally. The classical presentation of herpes zoster (Karthikeya Patil, Mahima Patil 2004 JSS Dental College, Mysore)

involvement. The vesicles turn to scales in one week and healing takes place in 2-3 weeks (Fig. 13.4).

Diagnosis

Based on the clinical symptoms and the serological procedures described under primary herpes simplex infection.

Complications

Generalized herpes zoster with involvement of internal organs and post herpetic neuraligia. Post herpetic neuralgia has been shown to be caused by inflammation and fibrosis of the affected nerve and may cause severe pain months or years after the skin lesions have healed.

Oral Findings

The trigeminal nerve is the most commonly involved but lesions of the eye and forehead caused by involvement of the 1st division are much more common than oral involvement.

Treatment

Treatment is symptomatic in uncomplicated cases of skin or mouth involvement.

Herne K et al (1996)[11] have stated that the nucleoside analogues, acyclovir, famciclovir and valaciclovir, appear to shorten the duration of Post Herpetic Neuralgia (PHN) to a similar degree. Acyclovir is taken 5 times daily for 7 days, while famciclovir is taken 3 times daily for 7 days. Valaciclovir has not only been proved to be more efficient than acyclovir (i.e. 3 times daily administration) but also more effective in shortening the duration of PHN.

Whitley RJ; Weiss H et al (1996)[12] conducted randomized and controlled studies on 208 immunocompetent patients above the age of 50. A total of 15 university hospitals was enrolled for the study. They used following doses — Acyclovir 800 mg 5 times a day for 21 days and prednisone for 21 days in 60 mg per day for first 7 days, 30 mg/day for next 7 days, and 15 mg/ day for last 7 days. Suitable matched placebo was also included in the study. Combined acyclovir and prednisone therapy can improve quality of life in older age group patients who are immunocompetent.

Jackson JL et al (1997)[7] have mentioned that upto 15 percent of HZ patients experience some kind of post herpetic neuralgia. They performed meta analysis of 30 clinical trials of oral acyclovir in immunocompetent adults and found that treatment of herpes zoster with 800 mg X 5 times/d of oral acyclovir within 72 hours of rash onset may reduce the incidence of post herpetic neuralgia.

Kubeyinje EP (1997)[8] compared HZ treatment of 42 healthy young adults with acyclovir of the dose 800 mg 5 times daily for 7 days with 40 similar healthy patients suffering from HZ but without using acyclovir. He found no statistical difference in the post operative pain and healing time of the 2 groups. He opined that in developing country like India it would be imprudent to give oral acyclovir which is so expensive, to immunocompetent patients only.

Bareggi SR et al (1998)[9] studied[11] HZ and post HZ neuralgia patients for effect of local acetylsalicylic acid/ Diethylether (ADE) and systemic acetylsalicylic acid. (ASA). They concluded that local application of ADE gave superior pain relief both for primary lesions and post herpetic neuralgia.

Gnann JW Jr; Crumpacker CS et al (1998)[10]. In their controlled case studies found 40 mg Sorivudine once daily for 10 days is an effective drug for the treatment of herpes zoster in HIV-infected patients and results in better healing with less pain as compared to acyclovir therapy.

Medical treatment by Phenytoin or Tegretol controls pain in some cases while alcohol, block or nerve section may be necessary for intractable pain in case of neuralgia.

Ernst ME et al (1998)[13] has warned against indiscriminate use of corticosteroids for prevention of PHN. They mention that although systemic dissemination of virus is a possibility in clinics it is rarely actually encountered. The older patients chosen for this therapy must be healthy in all other respects with no trace of immunocompromise.

Herpangina

Etiology

Caused by Coxsackie A 4 virus in majority of cases but types A1 to A10 and A15 to A22 have also been implicated.

Clinical Manifestations

Generalized symptoms of fever, chills and anorexia occur. The patient will also complain of sore throat, dysphagia and sore mouth. Bilateral discrete small vesicles most commonly involving the posterior pharynx, tonsils, faucial pillars and soft palate occurs. Lesions are found less frequently in the buccal mucosa, tongue and hard palate. Within 24 to 48 hours the vesicles rupture forming small 1 to 2 mm ulcers. The disease is usually mild and heals without treatment within a week.

Difference between IRHS and HRP

Herpangina HRP	*Herpes simplex IRHS*
1. Occurs in epidemics	Does not occur in epidemics
2. Milder than IRHS	
3. Posterior portion is involved (of oral cavity)	Anterior portion is involved
4. Limited distribution of vesicles	Widespread involvement
5. Vesicles are smaller than IRHS	

Laboratory Findings

A smear taken from the base of the fresh vesicle and stained with Giemsa will not show ballooning degeneration or multinucleated cells.

Treatment

It is a self limiting disease and the treatment is supportive including proper hydration and topical anesthetic, when eating or swallowing is difficult, and fever control.

Hand-foot and Mouth Disease

Etiology

Caused by infection with Coxsackie's A16 virus.

Clinical Manifestation

Low-grade fever, oral vesicles and non-pruritic macules, papules and vesicles characterize the disease, particularly on the extensor surfaces of the hands and feet. The oral lesions are more extensive than those described for herpangina and lesions of the hard palate, tongue and buccal mucosa are common.

Treatment

Same as Herpangina.

Hoof and Mouth Disease

This disease is more likely to be seen in the village families who have cattle and those patients who graze, or milk the cattle tend to get the infection from them. Hoof and mouth disease is a viral disease more commonly seen in cattle and other cloven hoofed animals and is occasionally transmitted to humans. Following an incubation period of 2 to 5 days the patient complains of fever, headache and excessive salivation. These symptoms are followed by vesicles in various areas of the mouth and in the palm of the hand, soles of the feet and the interdigital surfaces of the fingers and toes. The vesicles enlarge and rupture leaving irregular eroded areas. The acute phase with fever persists for a week or more, after which the lesions gradually heal during an additional 2-week period. Treatment is largely symptomatic.

AUTOIMMUNE GROUP

Erythema Multiforme (EM)

EM is an acute disease of the skin and mucous membrane that may cause several types of skin lesions, hence the name multiforme. The oral lesions rapidly rupture and are often the only presenting lesions.

Etiology

Erythema multiforme is mediated by deposition of immune complexes in the superficial microvasculature of the skin and mucosa. Factors that trigger the immune complex vasculitis include food allergy, drug allergy, reactions to microorganisms and radiotherapy. Erythema multiforme reactions have been related to a wide variety of bacterial, fungal, and viral infections. Episodes of erythema multiforme have been related to leiomyoma of the stomach and uterus as well as fibroma of the ovary, Addison's disease, sarcoidosis, Crohn's disease of the bowel and carcinoma. Just over 50 percent of the cases are of unknown etiology with stress or emotional factors as the second largest category.

Clinical Manifestations

General Findings: It has an acute or explosive onset, symptoms such as fever and malaise occur in severe cases. Patients have extensive lesions of the skin and mucosa.

EM simplex is the least severe form and is characterized by macules and papules 0.5-2 cm in diameter appearing in symmetrical distribution. The vesiculobullous from of the disease is more severe and can cause extensive sloughing of the skin leading to severe disability or occasional death caused by secondary infection or fluid and electrolyte imbalance. The most common cutaneous areas involved are the hands, feet and the extensor surfaces of the elbows and knees. The face and neck are commonly involved. The pathognomonic ***target or iris*** lesion should be looked for.

Oral Lesions

Oral lesions commonly appear with the skin lesions. It starts as bullae on an erythematous base but intact bullae are rarely seen as they break rapidly into irregular bleeding ulcers. Lips are prominently involved and gingival involvement is rare. In full blown clinical cases the lips are extensively eroded and large portions of the oral mucosa are denuded of epithelium. The patient cannot eat or even swallow and drools blood tinged saliva. Healing occurs within 2 weeks in majority of cases but in some severe manifestations the patient may suffer for several weeks.

Treatment

Only supportive treatment. Oral hygiene is improved. Topical anesthetic mouthwashes are used. Soft or liquid diet is given and I.V. fluid to prevent electrolyte imbalance. Severe erythema multiforme may be treated with a short course of Steroids. An initial dose of 30mg/day of prednisolone is given for several days, which is slowly tapered.

Stevens-Johnson Syndrome (SJ Syndrome)

Generalized vesiculo bullous EM of the skin, mouth, eyes and genitals is Stevens Johnson syndrome.

Acute Epidermal Necrolysis

It is characterised by large, transient bullae and peeling of the skin, often with mucous membrane involvement. Two clinically similar forms of acute epidermal necrolysis are seen: Lyells syndrome (Toxic Epidermal Necrolysis) and Ritters disease (Staphylococcal scalded skin syndromes).

Lyells syndrome is most often seen in adults, drug related, with several features in common with the erythema multiforme.

Ritters disease affects children primarily and is associated with coagulase positive staphylococci.

UNKNOWN ORIGIN

Aphthous Ulcers-Recurrent Aphthous Stomatitis

Recurrent aphthous stomatitis is a disease of unknown etiology characterized by ulcers of the oral mucosa, which are variable in frequency and recurrent in nature. On basis of clinical features, three types are identified; Minor, Major and Herpetiform.

Most common form of RAU is the Minor aphthous stomatitis. It begins in childhood or adolescence. It begins as irritation to the loosely attached mucosa and then one or many small i.e. 2-4 mm ovoid ulcers form. They have erythematous margin. Keratinized mucosa of hard palate, dorsum of the tongue or gingival is usually spared. Ulcers heal uneventfully within 5 to 10 days. Sometimes secondary infection prolongs the agony (Fig. 13.7).

About 1 in 10 patients of RAU are seen to suffer bigger ulcers, greater than 5 mm to even 1.5 cms, which take longer to heal, sometimes upto 21 days and heal by scarification. These are termed as Major aphthous ulcers. These are also termed as PMNR or Periadenitis Mucosa Necrotica Recurrens.

Another 10 percent of RAU patients present with Herpeteform aphthous ulceration. This type has a female predisposition and onset around third decade of life. 1mm, discrete, multiple, (15-90) ulcers characterize this type. Often these ulcers coalesce to form larger irregular ulcers, which are extremely painful.

Major aphthous ulcers also refered as Suttons disease or Periadenitis Mucosa Necrotica Reccurance (PMNR) (Fig. 13.6). They are larger aphthous ulcers (> 1cm) which

lasts longer than a week or even months. This is usually associated with other systemic disorders like Crohns disease, Behcets syndrome etc.[21] (Fig. 13.5).

RAU associated with syndrome

Behcet's disease is a multisystemic disorder. It has three components, RAU, Recurrent genital ulcers and Eye lesions. Systemic involvement of joints, CNS and Psychiatric disturbances predominate. Japanese and Eastern Mediterranean populations have higher incidence and it has a positive HLA association.

Salvatore Gulli, Carlo Arrigo, Loredana Bocchino et al[20] have reported of remission of Behcet's disease with anti-tumor necrosis factor monoclonal antibody therapy.

The inheritance of recurrent aphthous ulcers was investigated by Miller et al [1] 1980 and they proposed that genetic basis in certain families was very strong specially when both the parents were suffering from RAU.

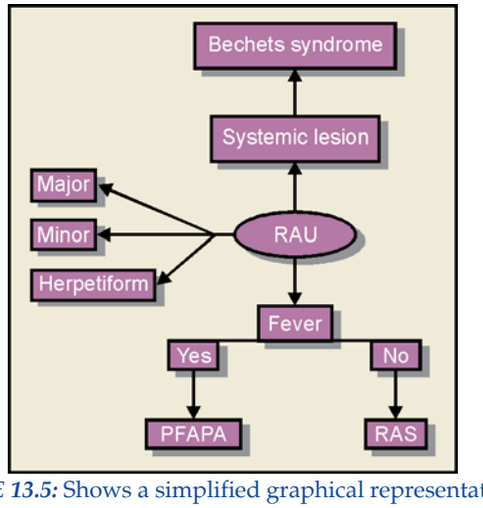


FIGURE 13.5: Shows a simplified graphical representation for classification of RAU

Even though number of theories exist regarding the etiology following clinical pointers are of importance while managing the case of RAU.

- A complete hemogram is recommended.
- History of psychologic stress/situational problems like exams, marriage, divorce, etc.
- Recent change in toothpaste, dentures, mouthwashes would point towards allergic etiology.
- Diet history is elicited for allergy to dietary components; in India spices appear to precipitate RAU in a larger number of patients.
- GI tract disturbances which wipe out the commensals, lead to B complex deficiency.
- Systemic diseases like Crohn's disease, Anemia, Gluten enteropathy, Cyclic neutropenia, and Agranulocytosis are some of the factors to be considered in the differential diagnosis.

FIGURE 13.6: Major Aphthous Ulcer in a 18-year-old female since two weeks. No apparent risk factor was elucidated in this patient. Orabase gave some relief (Nillofer S, Prasanna K, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

FIGURES 13.7A and B: Minor Aphthous Ulcers in two patients both of whom were appearing for professional examination in two weeks time. Minor RAU is often seen in patients with stress, B Comlplex deficiency or other forms of immune depression (Nillofer S, Prasanna K, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

Refer Table 13.1 for differentiation between RAU and Intraoral Recurrent Herpetic Stomatitis (IRHS).

Treatment

It should be aimed at identifying the influences mentioned above paying particular attention to trauma, deficiency of iron and folate, allergy to food, and stress factors.

Following medications in combinations may be tried by the clinicians:

- B complex capsules (Becosules®, Stresscaps® Cobadex® etc.
- Anxiolytic agents like Dizepam (Valium®-2mg, 5mg, 10mg; Aanxol® 2mg, and in severe cases intralesional steroids (Betnesol® 0.5mg per ml.) can be given.
- Local antinfectives like Chlorhexidine (Hexidine®) mouthwash, Tetracycline mouthwash (Terramycin®)
- Local pain controlling agents - Mucopain®, Lignocaine viscous®, Hexigel®

In case RAU persists and becomes aggravated it is better for the dental surgeon to refer the case to a bigger hospital, which will have more sensitive diagnostic modalities.

Major apthous ulcers are to be treated by excision with primary closure, cryosurgery or intralesional corticosteroids.

Barnadas MA et al[15] 1997 studied in 80 patients with recurrent oral ulcerations (ROU) levels of Iron, folic acid and vitamin B12 and the results were compared with a control group of 29 patients with different oral diseases. Patients with recurrent oral ulcerations have more frequently iron, folic acid and vitamin B12 deficiencies than those with other diseases of oral mucosa.

Chandrasekhar J[14] 1999 assessed twenty-four patients of RAU and treated them with a 4-week period of treatment with oxypentifylline (400 mg administered orally 3 times daily). The encouraging results of this study support the use of oxypentifylline in difficult cases of recurrent oral aphthous ulcers.

Chandrashekar T[22](1996) conducted a study comprising of 297 students 109 from professional and 188 from non professional college for prevalence of recurrent aphthous ulcers. He used general health questionnaire, state and trait anxiety and Bech's depression inventory. The prevelance of RAU in professional students was 59% compared to 36.6% in non professional college students. Examination was a strong precipitating factor with 65% of students complaining of RAU during exams compared to 7.6% in non professional students. GHQ, state and trait anxiety values showed a positive co relation to ulcers except in professional college females who showed a reversal. Other precipitating factors included: food stuff (sea food, cheese, spicy conditioning), pre-menstrual tension and allergy to mouth wash or tooth paste.

Bailoor DN and Nillofer S[23](2004) conducted a study to evaluate the relationship of stress to occurrence of RAU. They examined professional students of 3rd and 2nd year BDS, 6 months before and during university exams. During the initial evaluation 33.8% of them had RAU 61.3% developed RAU during exams. They used General health questionnaire. Hospital anxiety and depression scale and Dental environmental stress questionnaire, all these variable significantly increased during exams in those students who developed RAU during exams. A positive

Table 13.1: Weathers and Griffin 1970[3] have given a very clear basis to the differentiation between the RAU and IRHS

Characteristics	*RAU*	*IRHS*
AGE	Wide range	More common in middle and older
SITE	Freely movable mucosa (Non keratinized) lips, buccal mucosa, tongue, mucobuccal fold, floor mouth, soft palate.	Fixed mucosa. tightly bound to periosteum keratinized, hard palate, gingiva, alveolar region.
INITIAL LESIONS	Reddish macular or lesions undergoes certain blanching followed by necrosis and ulceration	Cluster of small discrete gray or white vesicles without red erythematous halo- vesicles quickly rupture forming1mm or less in diameter ulcers.
MATURE LESIONS	Shallow ulcer, 0.5-2 or 3 cm in diameter. Yellow necrotic center regular border, constant erythe mate's halo.	Shallow ulcer, no longer than 0.5 cm in diameter, multiple, coalesce to form large ulcers
NUMBER	Usually occur singly, or two or three widely present	Usually several small punctuated, ulcers in clusters, localized, are regular

association was also observed to family history of ulcers in students with frequent recurring episodes of RAU.

Mouth wash abuse ulcers: Moghadam BKH et al (1999)[16] has reported a case of over the counter mouthwash causing extensive mucosal ulceration in a 48-year-old female.

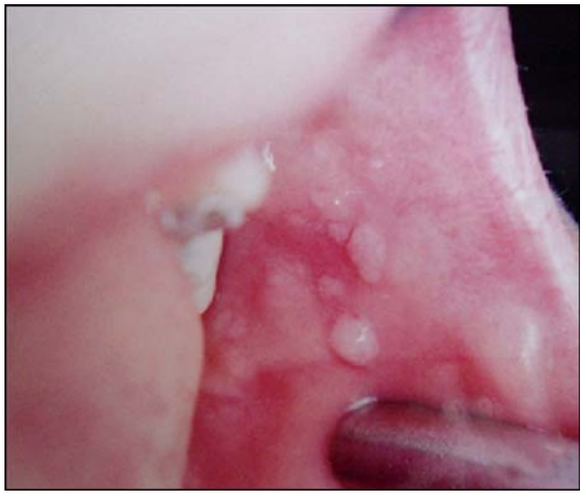
Kontogiannis and R J Powell (2000)[19] state that Behçet's disease is characterized by oral and genital ulceration, uveitis, skin manifestations, arthritis and a tendency to thrombosis. The underlying mechanism seems to be systemic vasculitis affecting venules. It has a worldwide distribution but is prevalent more in Japan, the Middle East, and some Mediterranean countries.

International diagnostic criteria have been proposed, however, diagnosis can be problematical. There is no single test, which is diagnostic, but the concept of pattern recognition helps in planning for correct diagnosis and therapy. Systemic steroids are the mainstay in combination with a number of drugs.

Pemphigus Vulgaris

It is a potentially fatal bullous disease of the skin and mucosa. It is an autoimmune disease in which antibody against intercellular substance of the epithelium acting with complement causes loss of cell-to-cell adhesion resulting in acantholysis (Fig. 13.10).

FIGURE 13.8: Figure showing Desquamative areas on the gingiva of a 32-year-old female patient who also had blisters like lesions on the skin also had hyperpigmented areas on the legs and arms. She was under therapy for Pemphigus vulgaris. The characteristic Nikolsky's sign was positive in her (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)



FIGURES 13.9A to C: Showing Skin and Oral manifestation of Pemphigus Vulgaris (Karthikeya Patil, Mahima Patil 2004 JSS Dental College Mysore)

Four variations of pemphigus: *Pemphigus vulgris, pemphigus vegetans, pemphigus foliaceus, and pemphigus erythematosus.*

Pemphigus vulgaris is the most common form- (80%) IgG type of auto-immunoglobulins cause seperation of cells by destruction of the intercellular substance. Pemphigus has also been reported to coexist with other autoimmine diseases particularly myasthenia gravis, patients with thymoma have a higher incidence of pemphigus. Several cases of pemphigus were reported which were produced by drugs like penicillamine used in the treatment of Wilson's disease and rheumatoid arthritis.

Clinical Manifestation

Lesions result from the destruction of intercellular substance in the prickle cell layer. This causes the upper layers of epithelium to pull away from the basal cell layer resulting in the acantholysis. The classical lesion of pemphigus is thin walled bullae arising on normal skin or mucosa. The bulla rapidly breaks but continues to extend peripherally, leaving large areas of denuded skin; pressure on an apparent normal area will result in the formation of a new lesion. This is called the *Nikolsky's sign* and results from the upper layer of the skin pulling away from the basal layer (Figs 13.8 and 13.9).

Oral Manifestation

Eighty to ninety percent develop oral lesions sometimes during the course of the disease and in 60% of the cases oral lesions appear first. The oral lesion may begin as the classical bullae on a non-inflamed base. Most commonly it starts on the buccal mucosa, often areas of trauma along the occlusal line appear. The palate and gingiva are other areas of involvement. A thin layer of epithelium peels away leaving a denuded surface. The edge extends peripherally leading to involve large portions of the oral mucosa.

Diagnosis

History helps differentiate from acute viral infection or Erythema multiforme. Lesions of pemphigus are shallow, irregular and have a detached epithelium on the periphery.

Lab Test

Biopsy must be taken from the advancing edge of the lesion where areas of characteristic acantholysis can be obtained. Cytology as described by Tzanck. A fresh lesion is scraped with the end of a scapula; material is spread on two slides, the slides, are fixed in methyl alcohol and stained with Giemsa or Papanicolane stain. On a positive smear, many separate acantholytic, rounded, epithelial cells will be seen with large deeple staining nuclei and prominent nucleoli.(Tzank cells)

Indirect Immunofluorescent antibody tests have been described: In this serum from a patient with bullous disease is placed over a prepared slide which is then overlaid with fluorescent tagged antihuman gamma globulin. Patients with pemphigus vulgaris will have antibodies against intercellular substance that will show up under a fluorescent miscroscope. The titer of the antibody has been directly related to the level of the clinical disease. Lab changes include leukocytosis, Eosinophilia and increases ESR.

Treatment

The mainstay of the treatment remains systemic corticosteroids—100 mg/daily of prednisolone initially, tapered later. It is best to involve an oral medicine specialist in a hospital setting instead of attempting treatments in dental clinic alone.

Bullous Pemphigoid

It occurs chiefly in children under 5 years of age and adults over 60. It is self-limited and rarely lasts over 5 years. In pemphigoid, the initial defect is not intra epithelial as in pemphigus vulgaris but is rather subepithelial in the region of the basement membrane. There is no acantholysis and no Nikolsky's sign being positive (Fig. 13.10). The disease is rarely life threatening because the bullae do not extend at the periphery to form large denuded areas as in pemphigus. The lesions remain localized and heal spontaneously. Etiology is unknown but circulating antibodies against a basement membrane zone antigen have been detected. No sexual or racial predisposition is seen.

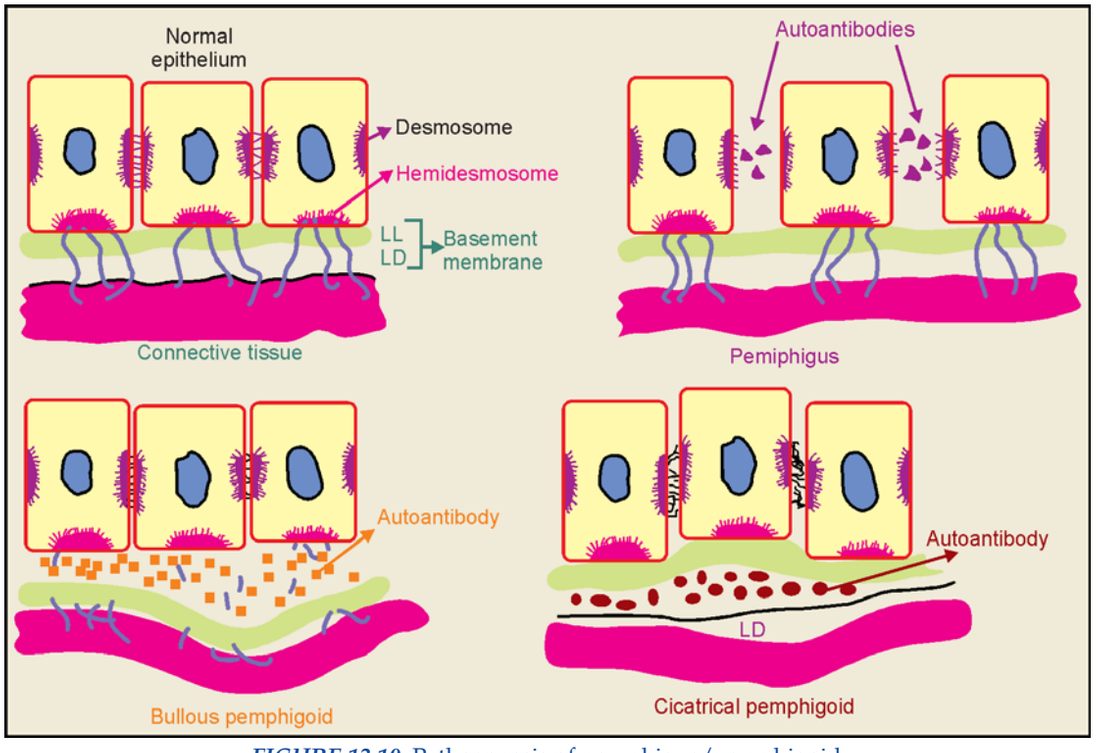


FIGURE 13.10: Pathogenesis of pemphigus/pemphigoid

Oral Manifestation

Oral involvement is less common than in pemphigus and is less likely to occur before the skin lesions, which are often relatively mild. Oral lesions most commonly occur in the buccal mucosa. The oral lesions of pemphigoid are smaller, form more slowly, and less painful than those of vulgaris. Extensive labial involvement seen in pemphigus is not present. The gingival lesions consist of generalized edema, imflammation, and desquamation with localized areas of discrete vesicle formation.

Diagnosis

Biopsy:Histologically will show subepithelial bulla formation in contrast to the intra epithelial lesions seen in pemphigus.

Indirect Immunofluoroscence antibody testing will demonstrate circulating IgG antibody against basement membrane antigens. Tzanck smears are negative for acantholytic cells. The direct immunofluoroscent test is more reliable in bullous pemphgoid as well as pemphigus and a biopsy specimen should be taken for direct immunofluroscent testing of IgG and complement whenever the diagnosis of bullous pemphigioid is suspected. Positive specimens will demonstrate IgG and complement in the basement membrane zone.

Treatment

Systemic corticosteroids are usually necessary although the doses are lower and given for a short duration.

Cicatricial Pemphigoid

Also called benign mucous pemphigoid. It is a chronic disease and chiefly occurs in patients over 50 years of age. The lesions are sub epithelial vesicles that occur on any mucosal surface and may lead to scarring of the affected region. This scarring is most dangerous when the eyes are involved. Adhesions may develop between the bulbar and palpebral conjuctiva (Symblepheron) and corneal damage results. Blindness occurs in 15 percent of the patients. Involvement of the esophagus and trachea leads to strictures and difficulty in swallowing. Skin involvement occurs in 25 percent of the patients (Fig. 13.10).

Diagnosis

Immunofluorescent testing is used to distinguish cicatrical pemphigoid from bullous pemphigoid because antibodies against the basement membrane zone are not positive in cicatricial pemphigoid. Using the direct immunofluorescent technique biopsy specimens taken from cicatricial pemphigoid patients will demonstrate positive fluorescence for immunoglobulin and complement in the basement membrane zone in 50% to 80% of the patients.

Oral Manifestations

Oral lesions are the most common finding in cicatricial pemphigoid and the mouth may be the only site involved. The chance of observing intact vesicles is greater in cicatricial pemphigoid than in pemphigus because the lesions are thicker walled, being subepithelial rather than intra epithelial. Gingival lesions have been described as a form of desquamative gingivitis. All cases of desquamative gingivitis should have cicatricial pemphigoid ruled out as a possible cause.

Erosive Lichen Planus

The oral lichen planus normally has small, delicate striae. When ulcerative form is seen then it is termed as erosive lichen planus. 27.6% of the LP cases are said to be erosive as per the study of Banoczy J (1982).[17] This type of LP has the highest malignant transformation rate. The actual figures of premalignant transformation appear to be varying with geographical variations. This rate in oral LP has been reported to be 0.3% by Gupta PC et al[18] 1980. As a private dental practitioner never attempt treatment of the Erosive LP. The primary responsibility should be perform a biopsy, and refer the patient with a proper histopathological report to a teaching dental hospital or oncology department.

CONCLUSION

When the vesicles appear in the oral cavity the clinician must ascertain whether the problem is of allergic, viral or autoimmune nature. When possible, serological tests, testing of the fluid of the vesicle and meticulous history will give a hint to the underlying cause. In many of the lesions discussed in this chapter the dermatologist's expertise will help the dental surgeon for a more comprehensive patient care.

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14 Pigmentation of the Oral and Perioral Tissues

INTRODUCTION

The word pigment means color or coloring agent. The normal color of the oral mucosa is varying from pale pink, to deep bluish purple to sometimes even blackish. The normal range of color depends on:

1. Melanogenesis and distribution of the melanin pigment
2. Keratinization
3. Depth of epithelialization
4. Vascularity

Melanin producing cells—the ***Melanocytes*** have their origin in the neural crest. These cells have been postulated to migrate to the basal cell layer of the epithelial layers. They are amoeba like in shape with various tubular process which transfer the pigment to the surrounding keratinocytes. The number of melanocytes is equal in fair skinned and dark skinned people only the level of melanogenesis varies. The melanin production is controlled by:

1. Sunlight
2. Hormones
3. Genetic constitution/Racial factors

CLASSIFICATION

Thibodeau EA et al (1997)[1] in their study used a narrow-band reflectance spectrophotometer to measure melanin and hemoglobin pigmentation in the lips and skin of individuals. They found it a useful tool for quantifying differences in melanin and hemoglobin pigmentation in

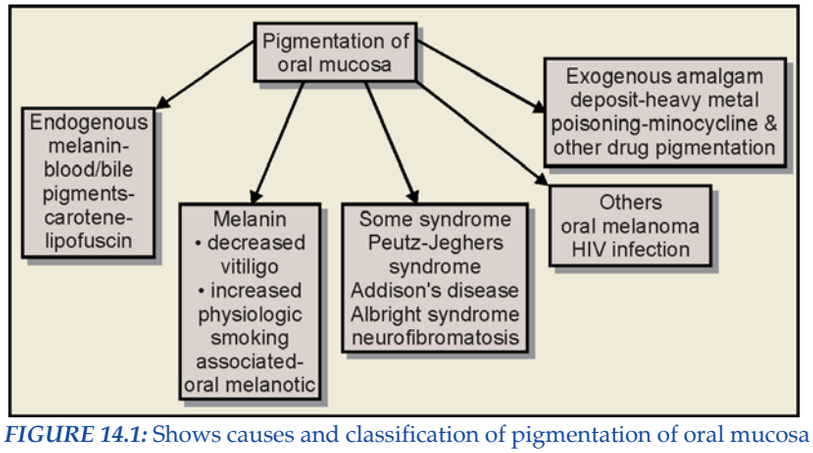


FIGURE 14.1: Shows causes and classification of pigmentation of oral mucosa

oral tissues. The following narrative continues on this points:

- Endogenous: Melanin—Blood/bile pigments-Carotene-Lipofuscin
- Exogenous: Deposited as such or formed as a reaction of a chemical of exogenous origin, like metal.
- Drug related pigmented lesions
- Some associated syndromes
- Miscellaneous lesions (Fig. 14.1)

ENDOGENOUS PIGMENTATION

Endogenous: originates from within the body.
Pigments originating endogenously are:-

- Melanin
- Blood and bile pigments
- Carotene
- Lipofuscin.

Melanin

It is derived from the Greek word 'Melas' meaning black. It is an insoluble polymer always bound to a protein. It is a brown-black pigment. Depending upon the amount of melanin the color varies from pale brown to black.

The melanocyte is believed to be derived from the neural crest during embroyonic development. It resides in the basal layer of the oral epithelium and has a multi-dentritic morphology. The release of melanosomes and the subsequent melanin granules into the epithelium results in the variable amount of pigmentation (Fig. 14.2). The nevus cells are morphologically different but functionally similar to the melanocytes. One school of thought says that they evolve side by side to the melanocytes from the neural crest but other one says that they are derived from the melanocytes later. Nevii cells are able to secrete melanin and cause pigmentation. The superficial ones appear brown, and deeper ones become black to very deep ones showing a bluish hue. It is not clear whether the melanocytes or nevus cells undergo malignant transformation in the malignant melanoma (Fig. 14.3).

The melanocytes are having the intracellular organelle the melanosome in which tyrosine is oxidized to dopa by the enzyme tyrosinase during which the melanin is formed as the end product. The production of melanocyte depends upon the hormone MSH from the anterior pituitary gland.

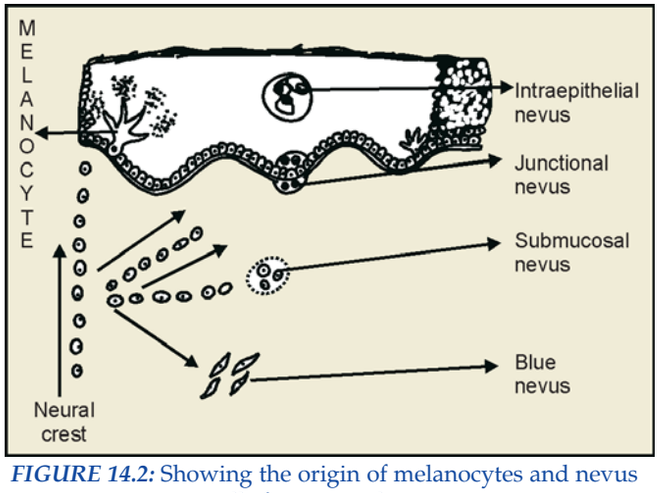
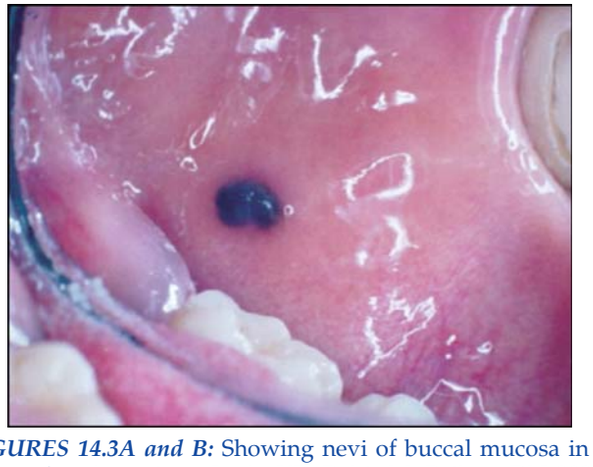


FIGURE 14.2: Showing the origin of melanocytes and nevus cells from neural crest



FIGURES 14.3A and B: Showing nevi of buccal mucosa in a young female patient (Leela KP, Rohit M, Bailoor DN 2003; Yenepoya Dental College and Hospital, Mangalore)

So any stimulation to neural crest cells or increase in the anterior pituitary function, for example, any endocrinal disturbances, result in increased production of melanocytes which in turn result in melanin pigmentation.

The melanosome with formed melanin goes to the hair follicles. These alterations can be classified into the following based on the cause.

Local and Systemic

Local-chronic irritation and inflammation stimulates the basal cell layer and causes melanin formation and increases the darkness of the mucous membranes. Sunlight stimulates the lower lip especially in tropics to form melanin (Fig. 14.4).

FIGURE 14.4: Shows mild pigmentation of the oral mucosa and this is due to natural variation (Omal PM, Been AK, Bailoor DN 2003 Yenepoya, Dental College and Hospital, Mangalore)

Systemic: Systemic alterations in melanin formation can be further subdivided into Hormonal, Chemical and Neurological origins

Hormonal: As MSH is formed in anterior pituitary gland any stimulation which causes anterior pituitary gland hypersecretion results in melanin formation. The following are the systemic conditions that cause endocrinal disturbances: **A.** Addison's disease **B.** Hyperfunction of pituitary glands. **C.** Pregnancy and female sex hormones. **D.** Polycystic fibrous dysplasia of bone (Albright's syndrome and Jaffe's type).

i. *Addison's disease*—This disease results from hypofunction of the adrenal gland. The clinical symptoms are weakness, nausea, and low blood pressure.

   Orally hyperpigmentaion of skin and mucous membrane will be noted. The hypofunction of adrenal gland causes increased secretion of ACTH. As the ACTH has melanocyte stimulating action MSH also increases and this causes the appearance of dark pigmented areas.

ii. *Hyperfunction of the pituitary gland:* This causes an increased secretion of the hormones ACTH and MSH. These hormones increase the melanization rate causing the abnormal pigmentation of the oral mucous membrane.

iii. *Pregnancy and female sex hormones:* Abnormal pigmentation of the circumoral tissues and nipples is seen in the 3rd trimester of pregnancy. This is termed as 'Chloasma gravidarum'. This also occurs with the usage of oral contraceptives containing large doses of sex hormones. In all these the function of the pituitary gland is going to increase which in turn causes increase in melanin formation.

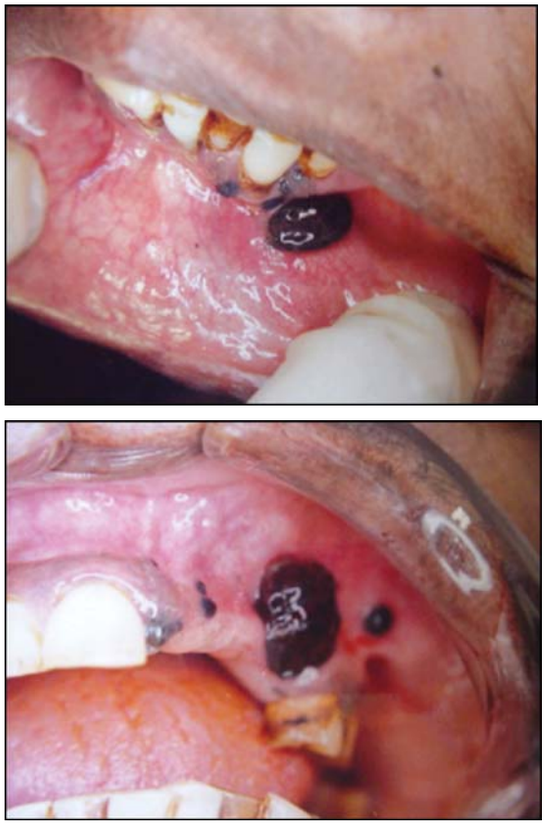
iv. *Polyostotic fibrous dysplasia of bone*: Fibrous dysplasia of the bone may be associated with the altered hormonal levels, the skin and oral mucous membranes have areas of small dark brown macular pigmentations. (Albright Syndrome).

Chemical: Certain drugs especially quinine and other antimalarial drugs have been on occasion shown to produce blue-black mucocutaneous pigmentations. These pigmentations result only with prolonged use of drugs and subside with their stoppage.

Neurologic: Neurofibromatosis or von Recklinghausen's disease of the nerves is a rare disorder of the neural tissues. Clinically it presents as multiple sessile tissues due to overgrowth of peripheral nerves. 'Café au lait' spots similar to fibrous dysplasia occur on skin and mucous membrane. As the melanocytes are of neural origin any alterations in the neural tissues as in this disease cause the stimulation of the cells and results in increased melanin pigmentations.

Developmental: These are basically due to alteration in the activity of the melanocytes. Blacks have pigment widely distributed in the oral mucosa. The common sites are the attached gingival and labial and buccal mucosa, which as termed as 'melanoplakia'. In Caucasians such macular lesions have been termed as oral melanotic macule (OMM). Congenital, developmental tumor like malformation of skin and mucous membrane termed as 'nevi' may be seen in some persons. It usually occurs in the skin but rarely in oral mucosa. They are usually flat, slightly raised, well-demarcated, discrete lesions. Treatment involves removal as there is suspected risk of malignant transformation.

Malignant Melanoma: This is a neoplasm of epidermal origin. It is a very unpredictable and deadly neoplasm, which is dark black in color. Mostly occurs in upper half of the face. Around 7 out of 10 intraoral tumors are in the palate (Fig. 14.5). They may develop de novo or in existing nevii. Clinically they may go through the radial growth phase, vertical growth phase and then into the ulcerated phase. Oral melanomas account for 1 percent of the total body tumors of this variety. Treatment is by surgical excision. Prognosis is not very good.



FIGURES 14.5A and B: Showing 55-year-old female patient with multiple pigmented papules on the maxillary and mandibular arches diagnosed as malignant melanoma (Patil K, Patil M, JSS Dental College, Mysore 2004

Blood Pigment and Bile Pigment (Hemoglobin Derivatives)

Hemoglobin is a vital pigment of the RBCs, which is essential for oxygen transport. Iron is its very important component. The hemoglobin is also seen in Kupffer cells of liver, spleen and bone marrow. Iron is released back into blood; the porphyrin portion of the Hb is converted into the bile pigment *BILIRUBIN*. The iron is stored in many forms. One such form is hemosiderin which is insoluble.

Hemochromatosis (Bronze Diabetes)

There is abnormal iron metabolism as a result of increased iron intake, absorption or parental transfusion of blood and iron containing fluids. The pigmentation results from deposition of abnormal amounts of iron and melanin. A characteristic bronze colored skin is present. Oral pigmentation consists of bluish gray pigmentation of hard palate and gingival tissues.

Jaundice

Jaundice which results by liver disorder causes improper metabolism of bile pigments associated with deposition of bile pigments in skin and oral mucous membrane. A yellowish hue is present in the oral mucous membrane.

Carotene

Carotenemia

This is a condition, which results from chronic excessive levels of carotene pigments in the tissues. This usually results from the long and continued consumption of large amounts of foods like carrots, egg yolk etc. Disturbances in metabolism of these foods to produce vitamin A may also increase carotene levels.

An orange yellow pigmentation of skin and mucous membrane occurs. It is similar to that due to bile pigments

except that the sclera is not involved. No treatment other than dietary modification is indicated.

Lipofuscin

It is an aging pigment, which will rarely affect the oral mucous membrane.

EXOGENOUS PIGMENTATION

The most common intraoral form of exogenous pigmentation is due to impregnation of foreign substances. Most common causes are:

- Accidental impregnation
- Iatrogenic impregnation
- Increased exogenous deposition.

Accidental Impregnations

In road accidents small bits of stone, gravel and sand get impregnated into the oral tissue. They, if not completely removal can cause discoloration. Charcoal containing dentifrices also produce black, permanent discoloration due to constant use.

Iatrogenic Impregnation

Amalgam tattoo: Small pieces of amalgam restoration can break off, impregnate into gingival, and oral tissues during fabrications and removal of restorations or extraction of teeth. It is not frequently seen now-a-days with increased care and facilities. The pigmentations are very frequently mistaken for melanin pigmentation; Purplish gray to blackish discrete pigmented lesions is seen.

Increased Exogenous Deposition

Heavy metal poisoning **(**Arsenic-Bismuth-Lead-Mercury): Heavy metals and its excess leading to oral manifestations is seen most commonly in the occupational hazards. Police personnel, hawkers plying their trade in the heavy traffic of big and small cities of India would seem to be more at risk. Some ayurvedic and alternative medicine tablets may contain heavy metals and such a history should be asked specially in some states in India a large number of patients may be going to alternate medicine practitioners.

Orally the gingival margin shows the linear black or gray line. One of the theories states that the reaction of hydrogen sulphide with heavy metals causes the bismuth or the lead line.

Chronic mercury intoxication was seen in the earlier days when manual manipulation of the mercury was common in the dental clinics. Earlier carelessness is replaced now with automatic trituration and well-ventilated clinics. The battery and chemical industries remain risk occupations for the heavy metal poisoning in India today.

Inner city slums of megacities like Mumbai, Kolkata, Delhi and Chennai remain high risk for the children who may get exposed to high levels of pollution from leaded gasoline and lead drinking pipes of outdated plumbing systems. Children under the age of 6 years appear to be in category of high risk because they are growing so rapidly and because they tend to put their hands or other objects into their mouths. Most of these kids stay in older buildings and come from low socioeconomic levels. Lead in the body is dangerous because it interferes with normal metabolism. It can interfere with the way the blood forming cells work, alter the way nerve cells signal each other and long-term effects can cause mental retardation.

Older homes still have surfaces painted with lead paint. Young children eat, chew, and suck on lead-painted surfaces they can reach, like window sills and railings. The little children may put toys, jewellery and printed matter that may have lead in their mouths. Dirt and dust sometimes have lead in them, as do the fumes and dust stirred up during home renovation and while sandblasting lead-painted buildings and bridges. Older homes and especially deteriorating and poorly kept older homes can be a threat for children. Particular jobs, like welding, radiator repair, making lead batteries, and demolition work can be especially hazardous to workers. Contaminated drinking water (lead pipes, solder, brass fixtures, valves can all leach lead), home health remedies which do not have standardized components and hobbies like making stained-glass windows all may lead to lead poisoning. Lead poisoning can cause learning disabilities, behavioral problems, and, at very high levels, seizures, coma, and even death.[13]

Recent report from Mangalore city in the Vijay Times[12] has highlighted that at least 70 percent of the children sampled, who were below 12 years of age showed a serum

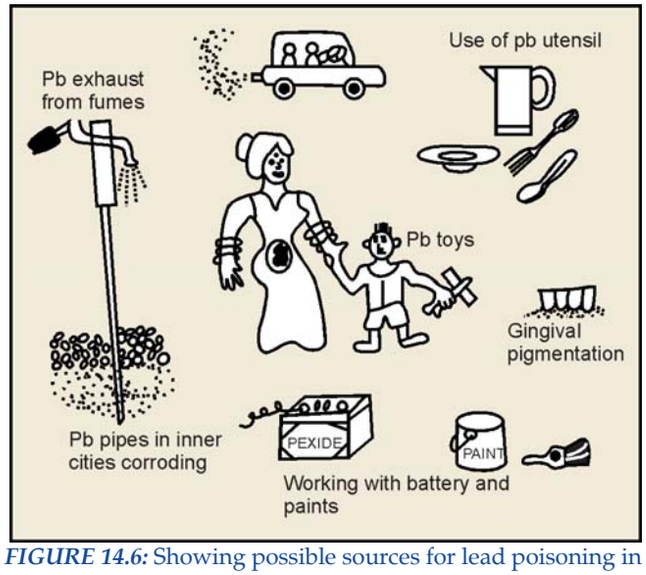


FIGURE 14.6: Showing possible sources for lead poisoning in the human body

level of 70 microgm/dl of lead as compared to the permissible level of 10 microgms/dl of lead. They identified that drinking water was the main culprit. The level determined in the drinking water was four to five times the safety limit as mentioned by WHO. Use of petrol with high lead content in vehicles, battery industry effluents, paint manufacturing industry pollution, paints used in schools and yellow colored paint in school buses, etc. were attributed as a cause for this poisoning. Local medical colleges have taken up the project for identifying and treating such children. Companies like Eureka Forbes and Filtrex International Ltd Singapore have developed a Sodium Aluminosilicate filter which is said to remove the lead in drinking water.

Choosing safer drinking water and a selection of multivitamins and calcium was recommended as treatment regimen. Nutritional counseling should include high iron and calcium, low-fat diet; frequent small meals (Fig. 14.6).

Table 14.1 shows what the oral medicine specialist must do depending on the blood lead levels in the child or the adult who shows the oral signs of lead poisoning.

Bismuthism: Bismuth is used for medicinal purpose. Despite widespread usage, toxic effects of bismuth are comparatively few. Medicinal use is for syphilis, non-specific diseases and suppositories. Proprietary drugs and pastes are the major causes of bismuthism. Clinically vague GIT disturbance, nausea, bloody diarrhea, gripe and jaundice is noted by chronic use. Bismuth lines can be demonstrated in bone radiologically.

Orally "bismuth line" blue black in color is noticed, in the marginal gingival especially in interdental, papillary and gingival tissues around erupting third molar and lingual gingiva of lower incisors. Gingivo-stomatitis

Table 14.1: Guidelines for evaluation of lead levels in the blood

Blood lead level (µg/dL)	*Suggested action by health care provider*
<10	No treatment required
10-14	Obtain a confirmatory venous BLL within 1 month; if still within this range, Provide education to decrease blood lead exposure Repeat BLL test within 3 months
15-19	Obtain a confirmatory venous BLL within 1 month; if still within this range, Change child's environment Provide education to decrease blood lead exposure and to decrease lead absorption Repeat BLL test within 2 months
20-44	Obtain a confirmatory venous BLL within 1 week; if still within this range, Conduct a complete medical history (including an environmental evaluation and nutritional assessment) and physical examination If BLL is >25 µg/dL, consider chelation (not currently recommended for BLLs <45 µg/dL), after consultation with clinicians experienced in lead toxicity treatment
45-69	Obtain a confirmatory venous BLL within 2 days Begin chelation therapy in consultation with clinicians experienced in lead toxicity therapy
>70	Hospitalize the patient and begin medical treatment immediately in hospital with poisoning specialist or MD with lead toxicity treatments

similar to ANUG is seen. Tongue is sore and enlarged. Diagnosis is from clinical finding by paper test performed to confirm gingival pigmentation and to eliminate suspicion due to stained tooth surface. Treatment is by establishing and maintaining oral hygiene and stoppage of the use of bismuth.

Lead Poisoning: Lead poisoning or ***Plumbism*** is very well known and widely documented. Excessive absorption of lead from exhausts, paint, occupational exposure leads to plumbism. Acute exposure is usually related to occupation. Alimentary canal and lungs are major sites for absorption.

In oral tissues lead affects by direct contact during ingestion or through salivary secretion, Oral symptoms are vague and overshadowed by the systemic symptoms. Probably the most important is the metallic taste. When exposure to lead is high and the oral hygiene is poor a leadline *Burtonian Line* is seen. It is gray black in color and is present along the gingival margin. It is probably due to formation of lead sulfide salt in the gingival crevice. Other oral signs include, pallor of tongue, poor muscle tone, and sunken face.

Diagnosis is by clinical and by laboratory tests. Treatment is elimination of toxicity followed by removal of lead from the body.

Mercurialism: Mercury is very easily absorbed. *Mercurialism or Ptyalism* develops as a result of occupational hazard, suicide attempts, or a result of self-medication.

Mathew A[14] suggests that recent reports have indicated that fresh water fish has been extensively contaminated with mercury. The pollution effluents from various industries like photographic chemical manufacture, battery production, etc. get released into water reservoirs and get converted into methyl mercury which enters into the food chain and causes neurological problems and learning deficits in growing children.

Systemically, GIT disturbances, headache, tremors of the fingers and of the tongue, is noticed. Renal symptoms indicate intoxication severely. In children it is collectively called as *Acrodynia*. Long continued exposure leads to permanent neurological changes ultimately in death. In oral cavity mercurialism causes increased flow and thick ropy saliva "*hot mouth*", itching sensation and metallic taste, a faint diffuse grayish pigmentation is seen. A mercury line similar to that of bismuth is seen. Oral ulcerations are severe. Tongue is enlarged; lymph nodes and salivary glands are also enlarged.

Diagnosis is from symptoms and laboratory tests. Treatment is by removing the cause, bed rest, treatment for renal damage and scrupulous oral hygiene.

Argyria: The use of silver containing medicines, chewing films over extended period, results in permanent discoloration of the skin and mucous membrane. The skin is slate gray, violet or cyanotic and with a metallic luster in severe cases. Pigmentation is diffusely distributed in oral tissues.

Arsenic: Usually due to chronic exposure from industries or a result of poisoning. Oral lesions are similar to that of mercurialism. Oral tissues are deep red painful and mouth is dry.

Copper Chromium and Zinc

Copper causes a bluish green discoloration or line on gingiva and teeth.

Chromium: on chronic exposure causes ulceration of oral tissues and mucosa may have an orange stain. Teeth are also stained. Cadmium presents similar symptoms.

Zinc poisoning causes congestion and suppuration of gingival tissues with a metallic taste. Bluish gray line is seen.

SIGNIFICANCE OF ORAL PIGMENTATION IN SYSTEMIC DISEASE

The pigmentation of oral mucous membrane is often associated with systemic disease. They can aid in diagnosis of the disease. Certain lesions like Nevi can become malignant and so constant observation in necessary. Racial and ethnic pigmentation can help in forensic Stomato-Odontology for identification of people.

Checking of serum levels, urine levels and neurological assessment all lead the dentist to the correct diagnosis. The physician may have to look at kidney and liver dysfunction for the correct diagnostic conclusion to be drawn.

DRUG RELATED PIGMENTED LESIONS

- Naproxen
- Minocycline
- Chloroquine
- Cis platinium
- Cyclophosphamide.

TI Gonzalo Garijo MA et al (1996)[5] reported that naproxen taken for dysmenorhea was the cause of pigmented lesions appearing periodically on the oral mucosa. They report that a 28-year-old woman was affected with vesicular lesions on the oral mucosa causing a burning sensation. She had noticed that these eruptions reappeared in the same location and related to menstruation (when she used to take naproxen sodium because of dysmenorrhea). Furthermore, pigmented sites became red-brown, elevated and itchy.

Cockings JM et al (1998)[6] have mentioned about the Minocycline causing the oral pigmentation as a side effect. Oral pigmentation usually involves discrete band occupying the central zone of the alveolar mucosa and palate or tongue, as with other sites, it may persist following withdrawal of the drug.

FIGURE 14.7: Shows a resident of Gadag district in Karnataka state known for its high fluoride levels in drinking water. The yellowish brown stain appears unsightly but there is no increase in the caries level in this patient (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

SOME SYNDROMES ASSOCIATED WITH ORAL AND PERIORAL PIGMENTATION

- PJ syndrome
- LH syndrome
- Addison's disease
- Albright syndrome
- Neurofibromatosis.

Peutz-Jeghers Syndrome (PJS)

The association between heredity, gastrointestinal polyposis, and mucocutaneous pigmentation in PJS was first recognized in 1921 by Peutz in a Dutch family.

Westerman AM et al (1999)[2] did mutation analysis in this family to test whether the recently identified LKB1 gene is indeed the PJS gene in this family, which has now been followedup for more than 78 years. DNA-mutation analysis was done in all available descendants. Clinical features in this family included gastrointestinal polyposis, mucocutaneous pigmentation, nasal polyposis, and rectal extrusion of polyps. The morbidity and mortality in this family suggest that PJS is not a benign disease. An inactivating germline mutation in the LKB1 gene is involved in the PJS phenotype in the original and oldest kindred known to be affected by PJS.

Hemminki A (1999)[3] mentions that PJS predisposes to cancer. The most common malignancies are small intestinal, colorectal, stomach and pancreatic adenocarcinomas. Other cancer types that probably occur in excess in PJS families include breast and uterine cervical cancer, as well as testicular and ovarian sex cord tumors. The relative risk of cancer may be as high as 18 times that of the general population. The causative factor was shown to be LKB1 gene mutation.

Yagmurdur MC et al (1998)[4] mention the usefulness of endoscopic polyp removal, which reduced both discomfort and the intestinal hemorrhage in PJS patient.

Laugier-Hunziker Syndrome (LH syndrome)

Idiopathic mucocutaenous pigmentation.

Mignogna MD et al (1999)[8] studied 12 patients in detail including the light microscopy and electron microscopy and established that this syndrome involving idiopathic mucocutaenous pigmentation showed no evidence of any type of malignancy occurring in this series.

Yamamoto O et al (1999)[10] mention about the first case report of an esophageal melanocytic macule which occurred in a patient with Laugier-Hunziker syndrome. Seoane Lest´on JM et al (1998)[9] in their analysis of 13 cases

of LH syndrome mentioned that the histopathological report revealed the presence of regular acanthosis, basal pigmentation without increase in the number of melanocytes and the presence of melanophages in subepidermal connective tissue have been the most constant histopathological characteristics. They have suggested the term "essential cutaneo-mucous hyperpigmentation" as synonym of Laugier-Hunziker disease.

Ferreira MJ et al (1999)[7] have reported the case of a 46-year-old Caucasian female presenting with mucocutaneous pigmentation on the lips, oral mucosa, hands, feet and nails, as well as on a psoriatic plaque. She was successfully treated with Q-switched Nd:Yag laser, with double frequency, for both the mucosal and cutaneous lesions.

MISCELLANEOUS LESIONS

That may be confused with pigmentations in oral cavity:

- **Varicosities:** of the tongue appear as distended veins on the ventral surface, in the oral cavity. It is more prominent in older individuals.
- **Hemangiomas:** sometimes give bluish, black lesions in tongue, oral mucosa and are associated with birthmarks. Superficial lesions are characteristic and deep-seated lesions require angiography for their diagnosis.

FIGURE 14.8: Shows brownish black stains on the teeth of a patient who is a habitual pan chewer and tobacco smoker. This causes heavy extrinsic stains on the teeth. However thorough scaling and polishing can easily remove it (Nillofer S, Prasanna K, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

- **Black Hairy Tongue:** Elongation of the filiform papillae result in this harmless condition reported to be 0.15 percent by Farman[11]
- **Pigmented Fibroma:** This reactive lesion is microscopically demonstrated since it clinically appears just like nevus.
- **Nevus** is a hamartoma containing melanocytes or nevus cells that occurs on the skin; intraoral occurrence rare. Four types intramucosal, junctional, compound and blue.

SUMMARY

The pigmented lesions of the oral cavity can confuse the uninitiated clinician. The lesions may vary from the benign macule the OMM to the most dreaded malignant melanoma. A good dentist can take help of the medical colleagues in complete assessment of these patients so that a rare diagnosis may not be missed.

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Rawal Y, Bailoor DN

15 Cysts of the Oral Regions

INTRODUCTION

The word CYST (SIST) is derived from the Greek word KYSTIS meaning a bladder. Cysts are unilocular or multilocular sack like lesions enclosed by a capsule. They may occur anywhere in the body. The head and neck region with the jaws in particular comprise one of the more common sites of occurrence. The higher frequency of cysts in the orofacial region could be attributed to the complex embryology and the development of teeth with the presence of varying amounts of residual epithelial islands, which are potentially capable of being involved in the development of cysts.

Cysts form a particularly peculiar pathology of the human body as in a majority of cases the cause is unidentifiable and only hypotheses are postulated to explain their pathogenesis.

Much variation exists in the epidemiology and no studies are known that conclusively state the occurrence pattern of cysts of orofacial region in India.

CLINICAL SIGNIFICANCE OF CYSTS OF OROFACIAL REGION

The diversity of clinical alteration that cysts of orofacial region bring about requires a thorough understanding of their varied nature.

The cysts, especially the intraosseous variety may be easily mistaken as benign tumors.

The cysts may assume large proportions causing much local bone destruction with expansion and consequent disfigurement. Much bone destruction may result in thinning of the bone with tendency to fracture under even physiological forces. The cysts may arise either due to trauma, as a result of inflammation and degeneration or retention. They may be congenital or acquired.

Further, cysts may get secondarily infected and lead to abscess formation, which may complicate into cellulitis, osteomylitis and concomitant sinus formation.

Cysts may also interfere with and alter the normal eruption pattern of teeth. Neoplasms, like the ameloblastoma and carcinomas like squamous cell and mucoepidermoid also arise from linings of the cysts.

The varied nature of cysts, therefore, requires a definitive and conclusive diagnosis as this recognition influences and alters the treatment modalities.

DEFINITION

Cysts are defined as pathologic cavities that may or may not be lined by epithelium and which contain a semisolid or fluid material.

Cysts that are lined by epithelium are termed as true cysts.

Cysts that do not possess an epithelium lining are called pseudocysts (false cysts).

PARTS OF A CYST

Macroscopic examination

The cysts have a central portion called as the LUMEN.

A leathery pouch like CYST MEMBRANE surrounds the lumen.

Microscopic examination: This reveals that the true cyst membrane is composed of (a) an epithelial lining, that faces the lumen and (b) a connective tissue capsule, which forms the outer perimeter of the cyst. Pseudocyst membrane does not show an epithelial lining and the luminal contents are in direct contact with the connective tissue capsule.

The Lumen

It is usually filled with a fluid matter. This fluid has been suggested to arise as a result of secretion, transudation/exudation and osmosis/dialysis. It would seem likely that it is a combination of all of these mechanisms that contributes to accumulation of the fluid, but that their relative importance will vary with the conditions prevailing in a particular cyst.

Cyst Fluid Composition

The presence of cholesterol crystals will impart a shimmering straw color to the fluid. These cholesterol crystals could be demonstrated by filtering the fluid through few layers of cotton gauze. When the fluid is filtered, the crystals are trapped by the fibers of the gauze and cloth shimmers when examined under oblique reflected light. The fluid could also be loaded on to a clean glass slide and covered with a cover glass and examined under a microscope for the presence of crystals. These crystals appear transparent, rectangular with one corner missing. They generally are seen in clumps over-lapping one another. The difference in the refractive index of the crystals from the fluid renders their boundaries dark and therefore visible.

Presence of keratin will produce a creamy yellow color. The consistency of the fluid varies from watery to an almost semisolid cheese like mass. Various components can influence the consistency, e.g. mucous secretions, cholesterol crystals and products of degeneration. The cyst fluid contains the following molecular constituents in variable amounts.

Blood Products

Presence of blood products would give a brownish hue.

A. Serum proteins:
   - Albumin
   - Alpha-globulins
   - Beta-globulins
   - Gamma-globulins.

B. Proteases and inhibitors
   - Collagenase
   - Alpha 2-macroglobulin
   - Alpha l-antitrypsin
   - Fibrinolysins (possibly)

C. Keratins and possible keratocyst-specific antigens

D. Glycosaminoglycans and proteoglycans:
   - Hyaluronic acid
   - Heparan sulfate
   - Dermatan sulfate
   - Chondroitin sulfate

E. Glycoproteins
   - Fructose containing
   - Hexose containing
   - Sialic acid containing

F. Lipids
   - Alpha 1 lipoprotein
   - Beta lipoprotein. (forming cholesterol)

CLASSIFICATION OF CYSTS OF THE HEAD AND NECK

A variety of classifications are available. They have been constructed by selecting certain features, which are common to a number of conditions, and grouping them together on the basis of their shared properties.

The embryological development, the tissue of origin or the histological features may be some groupings providing a basis for such classification: In this section, the authors proceed to present some well known classifications and seek to contribute their modification.

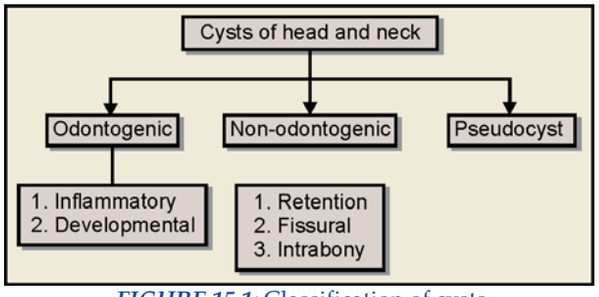


FIGURE 15.1: Classification of cysts

Gorlin's Classification (1970)

Odontogenic Cysts

- Dentigerous cyst
- Eruption cyst
- Gingival cyst of newborn
- Lateral periodontal and gingival cysts
- Keratinizing and calcifying odontogenic cysts
- Radicular cyst (Periapical cyst)
- Odontogenic keratocysts (Primordial cysts—Multiple Kerarocysts of the jaws)
- Multiple cutaneous nevoid basal cell carcinomas and skeletal anomalies.

Non-odontogenic and Fissural Cysts

- Globulomaxillary (Premaxillary-Maxillary) cyst
- Nasoalveolar (Nasolabial, Klestadts cyst)
- Nasopalatine (Median anterior maxillary) cyst
- Median mandibular cyst
- Anterior lingual cyst
- Dermoid and epidermoid cysts
- Palatal cyst of newborn infants.

Cysts of Neck, Oral Floor and Salivary Glands

- Thyroglossal duct cyst
- Lymphoepithelial (Branchial cleft) cyst
- Oral cyst with gastric or intestinal epithelium
- Salivary gland cyst
- Mucocele and ranula.

Pseudocysts of the Jaws

- Aneurysmal bone cyst
- Static (Developmental; latent Stafne's) bone cyst
- Traumatic (Hemorrhagic; solitary) bone cyst (Fig. 15.1).

The WHO Classification (1971)

Epithelial Cysts

Developmental

1. Odontogenic
   a. Primordia (Keratocyst)
   b. Gingival cyst
   c. Eruption cyst
   d. Dentigerous cyst
2. Nonodontogenic
   a. Nasoplatine duct (Incisive canal cyst)
   b. Globulomaxillary
   c. Nasolabial (Nasoalveolar) cyst.

Inflammatory: Radicular cyst

The following classification suggested comprehensively encompasses all benign cystic lesions of the head and neck region and is based on a combination of various features, for example, histology, tissue of origin, probable etiology, anatomic position, etc. The use of this particular classification and terminology does not imply that other classifications are not equally valid or useful.

Cysts of the Head and Neck (Rawal's classification)

True Cysts

Odontogenic

Inflammatory

1. Radicular (Root end periapical, dental) cyst
2. Paradental (Inflammatory, collateral) cyst
3. Inflammatory lateral
   Periodontal cyst (lateral radicular cyst)
   a. Follicular cyst
   b. Eruption cyst or eruption hematoma.
4. Residual cyst
5. Dental lamina (Gingival cyst)
   Cyst of the newborn
6. Gingival cyst of the adult.
7. Gorlin's (Keratinizing and/or calcifying epithelial odontogenic) cyst.

Developmental

1. Odontogenic keratocyst
2. Primordial cyst
3. Dentigerous cyst

4. Developmental lateral
   Periodontal cyst and the botryoid odontogenic cyst.

Nonodontogenic cysts
Retention cysts

- Mucous retention cyst.
- Sebaceous cyst.

Fissural/inclusion cysts

1. Globulomaxillary cyst
2. Maxillary median alveolar
3 Nasopalatine duct (Incisive canal, median anterior maxillary) cyst
4. Nasoalveolar (Nasolabial, klestadts cyst).
5. Thyroglossal tract cyst
6. Benign cervical lymphoepithelial cyst, branchial cleft cyst
7. Dermoid cyst
8. Heterotopic oral gastrointestinal cyst.

Intrabony/Soft tissue

1. Cyst of the palatine papilla
2. Epstein's pearls
3. Bohn's nodules
4. Median palatal cyst
5. Median mandibular cyst.

Pseudocysts (False cysts)

1. Aneurysmal bone cysts
2. Static bone cyst—Stafne's cyst
3. Traumatic cyst—Solitary bone cyst
4. Mucous extravasation cyst.

MECHANISM OF CYST GROWTH AND ENLARGEMENT

Following mechanisms are examined in relation to the cyst growth:

- Mechanism of cyst growth and enlargement are identical to all cysts of the head and neck.
- The mechanisms are different and characteristic for each type of cyst.
- The basic mechanism remains the same, but is altered by additional factors that determine the pathogenesis of each type of cyst.

The last assumption appears most plausible and acceptable.

The mechanisms involved in the growth of the pseudocysts will be dealt with individually with these cysts.

The retention cysts are usually a product of a blocked duct either due to formation of calcareous deposits or accumulation of debris comprising denuded epithelial cells and bacteria, particulate matter, vegetative matter, etc.

Ductal blockage leads to pooling of secretory product within the proximal aspect of the duct itself. This causes distention of the duct and the normal ductal epithelial lining forms the epithelial lining of the so called *retention cysts*. Alternatively, the discharging gland itself may distend due to synthesis of material with lack of ability to discharge due to duct blockage. The acinar cells or the intercalated duct cells would contribute to the formation of the epithelial lining.

Here, the growth pattern is dictated by the increased hydrostatic pressure due to secretion.

The growth and enlargement of *fissural* or *inclusion cysts* would be identical to the patterns of non-keratinizing odontogenic cysts. The various factors involved in the growth of the cysts are:

1. Retention of fluid within the cavity
2. Attraction of fluid into the cyst cavity
3. Raised internal hydrostatic pressure and osmotic pressure
4. Bone resorption with increased size of bone cavity.

Theories

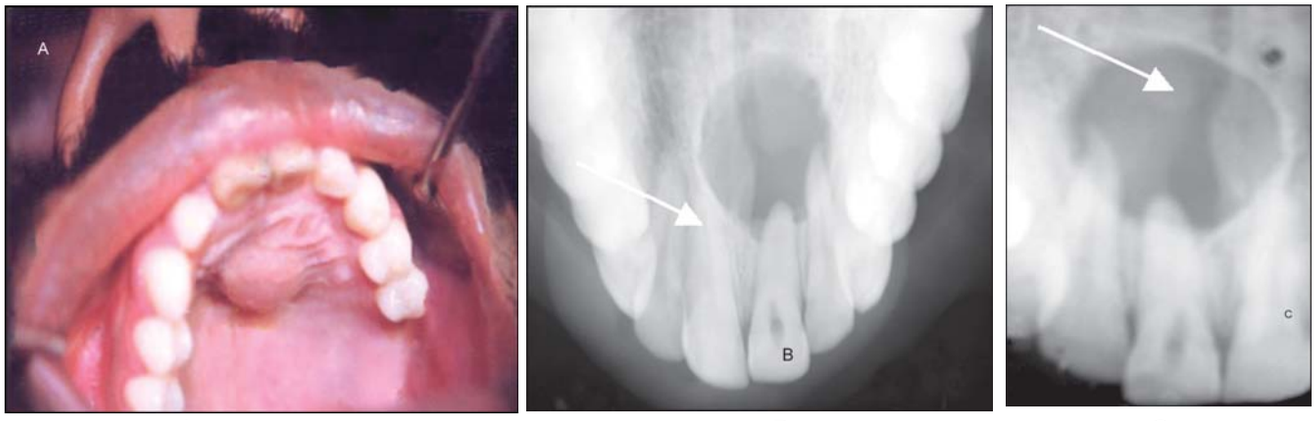
- The theories of cyst enlargement are as follows:
- Mural growth
- Peripheral cell division
- Accumulated contents.

Increased hydrostatic pressure:

- Secretion
- Transudation and exudation
- Dialysis and osmosis.

Pathogenesis

The growth of the non-keratinizing cysts, is circumferential unless influenced by resisting factors like a tooth which usually alters its shape. The growth of the odontogenic



FIGURES 15.2A to C: Shows 32-year-old male patient with a painless swelling in the palate since two months duration previous to which he had got hit while playing cricket (A) a large dome-shaped swelling with intact mucosa is seen in the mid palatine area measuring about 1.5 × 1 cm. (B) and (C) IOPA and occlusal radiographs showed a well defined radiolucent spherical area with apex of central Incisors. This was diagnosed as traumatic cyst (Nillofer S, Prasanna K, Bailoor DN 2003, Yenepoya Dental College, Mangalore, India)

keratocyst is uneven and guided more by proliferating epithelial cells burrowing into the bone at varying depths in different regions of the same lesion causing multiloculation. This factor plays an important role in the higher recurrence rate of the odontogenic keratocyst as explained later. Recently, the fibroblasts within the connective tissue capsule of many cysts, especially the radicular cyst, have been identified as being *myofibroblasts*. Their presence has raised a question of their ability in controlling the diameter of the lumen of the cyst (Figs 15.3 and 15.4).

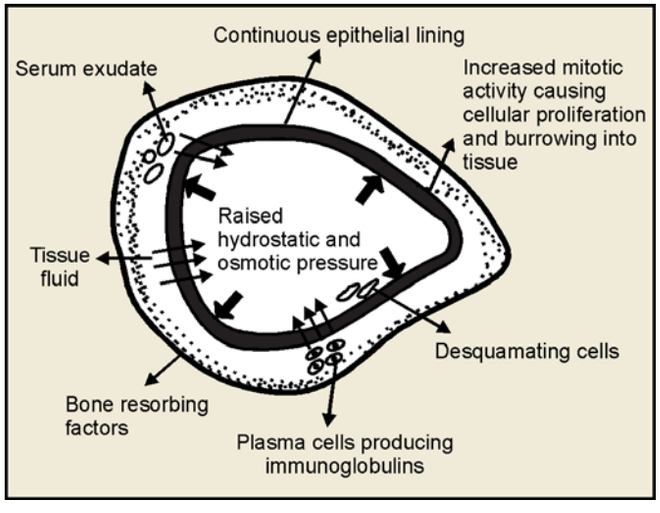


FIGURE 15.3: Factors involved in the growth and enlargement of odontogenic keratocysts

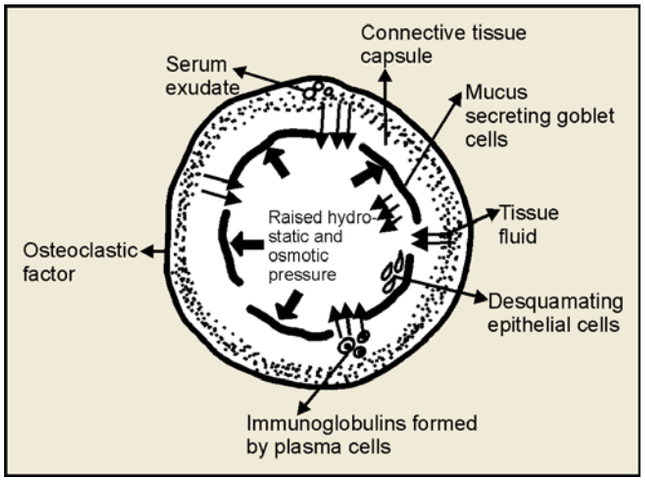


FIGURE 15.4: Factors involved in the growth and enlargement of nonkeratinizing odontogenic cysts

THE ODONTOGENIC CYSTS: THE INFLAMMATORY CYSTS

THE RADICULAR CYST

Synonyms

The root end cyst, periapical cyst, dental cyst, infected dental cyst.

Etiology

Dental caries, trauma causing pulp exposure, pulpal inflammation, pulp necrosis, periapical inflammation, periapical cyst, periapical granuloma.

The existence of a nearby area of inflammation stimulates epithelial cells to proliferate and to move towards the center of inflammation. The source of epithelium to the periapical region is from: The cell rests of Malassez. Respiratory epithlium of the maxillary sinus when the periapical lesion communicates with the sinus wall. Oral epithelium forms a fistulous tract. Or Oral epithelium proliferating apically from a periodontal pocket (Fig. 15.11).

Ramachandran Nair PN et al (1996)[3] Analyzed a total of 256 periapical lesions. Their results were 35 percent periapical abscess, 50 percent granuloma, and 15 percent cysts. The latter occurred in two categories, the apical true cysts and the apical pocket cysts.

Nair PN (1998)[2] mentions that the actual incidence of radicular cyst is only about 15 percent of all periapical lesions contrary to some other reports which suggest almost half of the periapical lesions are cysts. He believes that radicular cysts exist in two structurally distinct classes namely, those containing cavities completely enclosed in epithelial lining (periapical true cysts) and those containing epithelium-lined cavities that are open to the root canals (periapical pocket cysts). From a clinical point of view a periapical pocket cyst may heal after conventional root canal therapy whereas an apical true cyst is less likely to be resolved without surgical intervention.

Sanchis JM et al (1998)[4] studied the incidence of radicular cysts in 125 chronic periapical lesions in France. Histopathology revealed 18 radicular cysts (14.4%) and 107 lesions corresponding to chronic apical periodontitis (C.A.P.) or granulomas (85.6%). Cholesterol clefts, a fibrous capsule and presence of a cavity was seen in the cysts.

Clinical Features

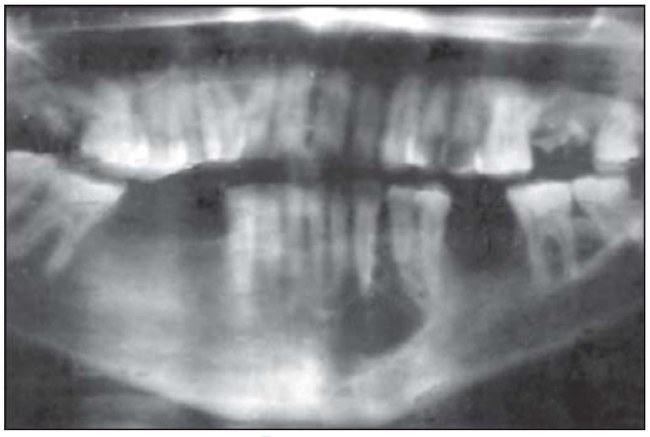
Majority of cases are asymptomatic. Tooth involved is carious but is seldom painful or tender on percussion. The cyst does not produce any noticeable bone expansion.

Pain is usually associated with an acute exacerbation and abscess formation. May result in sinus formation, osteomyelitis and cellulitis. Upon aspiration, the cyst yields a straw colored fluid that is rich in cholesterol crystals.

Radiographic Features

- Loss of laminadura at the periapical region.

A



B

FIGURES 15.5A and B: Shows(A) gingival swelling with obliteration of the buccal sulcus in a 42-year-old male Patient also had a history of trauma. Vitality testing with electric pulp tester showed negative results with the canine. (B) OPG of the same patient showed a large radiolucent Periapical area of the canine, the radiolucency was larger than 1 cm with well defined borders but with absence of a sclerotic margin indicative of infected radicular cyst (Nillofer S, Prasanna, Bailoor DN 2003, Yenepoya Dental College Hospital,Mangalore)

- Formation of a unilocular radiolucency.
- Periodontal membrance space becomes continuous with this radiolucency.
- Radiolucency is almost well circumscribed and well defined though the margins may be hazy.
- In many cases the margins may be sclerosed, suggesting a reactive bone deposition in response to a slow growing lesion. May show a mild degree of root resorption (See Figs 15.2, 15.5 to 15.10).

FIGURES 15.6A and B: 22-year-old female had trauma to the tooth while traveling on the bus six years ago. Central Incisor was discolored and painless. Radiograph revealed a large radiolucency suggestive of radicular cyst (due to traumatic reasons) (Nillofer S, Bailoor DN 2004 Yenepoya Dental College, Mangalore)

FIGURE 15.7: A 27-year-old female came with severe pain in the first mandibular molar . Radiograph revealed well demarcated lesion with this non vital tooth. Diagnosis radicular cyst (Prasanna K, Bailoor DN 2004 Yenepoya Dental College, Mangalore)

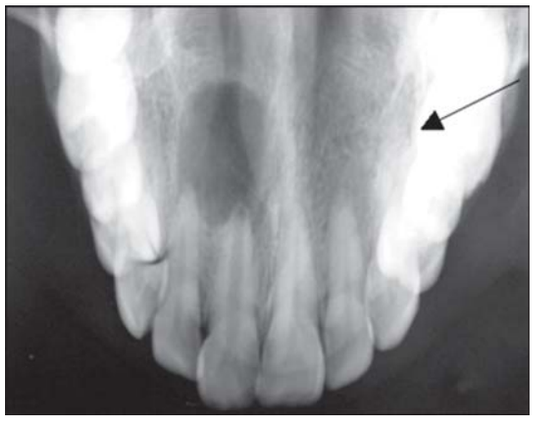
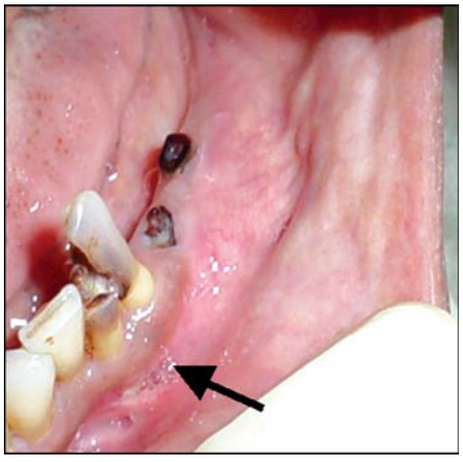


FIGURE 15.8: Shows well defined single unilocular radiolucency in the Periapical region of central incisors,indicative of radicular cyst (Beena K, Omal PM, Bailoor DN 2003, Yenepoya Dental College Hospital,Mangalore)

FIGURE 15.9: Shows maxillary Occlusal radiograph of radicular cyst with minimal buccal cortical plate expansion (Nillofer S, Prasanna K, Bailoor DN 2003, Yenepoya Dental College Hospital ,Mangalore)

Kizil Z and Energin K[5] from Istanbul reported in their study of 108 periapical lesions, the incidence, the relation of lesion size to the incidence, the diagnosis and the correspondence between radiographic and histopathologic diagnoses were investigated. It was established that the incidence of radicular cysts rose as the extent of the lesion increased and that the incidence of radicular cysts was 100 percent when the lesion size was 200 mm^2 or more. The X-ray findings were in correspondence with the histopathological diagnosis in only 66.6 percent of cases and it was concluded that radiography alone was insufficient to make a differential diagnosis of periapical lesions.



A

B

C

FIGURES 15.10A to C: Show a extraordinarily large radicular cyst in a 55-year-old female patient who came with the complaint of pain and swelling in her left lower jaw since 3 months. (A) Clinical picture shows root stumps with canine and deep carious lesion with lateral incisor of the left side. There was obliteration of the buccal sulcus extending posteriorly and elevation of floor of the mouth. (B) Occlusal radiograph shows expansion of the buccal cortical plate with thinning. (C) The massive unilocular, Scalloped cyst extending anterioposteriorly measuring around 7x 3 cm can be seen in this OPG (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College, Mangalore)

Treatment and Prognosis

If the periodontal condition permits, root canal therapy combined with endodontic surgery may be carried out. The cyst should be completely removed during the apicoectomy procedure. Prognosis is very good and recurrence rare.

Complications

Incomplete removal may result in recurrence.

Incomplete removal or failure of removal due to non-recognition of the condition, due to absence of any symptoms after tooth extraction results in the formation of a *residual cyst*.

The Paradental Cyst

Synonyms

Inflammatory collateral cyst.

Introduction

The paradental cyst is a relatively new entity. It is found only in association with a partially erupted mandibular third molar, where there has been a history of pericoronitis.

Pathogenesis

The evidence to suggest the possible origin of the epithelial lining is lacking. The position of the cyst in relation to the tooth would suggest the source to be, the cell rests of Malassez. The bifurcation areas of the third molars associated with this cyst show prominent enamel spurs, which would lead us to assume that the epithelial lining could be possibly derived from the reduced enamel epithelium overlying the partially erupted tooth.

Clinical Features

They may occur predominantly in a younger age group.

- Tooth involved is vital.
- The only symptoms may be associated with pericoronal inflammation.
- The cyst usually lies distal, distobuccal or mesiobuccal to the root of the partialy erupted tooth producing a slight swelling.
- Occasionally the cysts may be bilateral.

Radiographic Features

The involved tooth is partially erupted and is usually free of carious involvement.

- The cyst is usually placed distal or distobuccal to the involved tooth within the bone and rarely extension may be noted on the mesial aspect.
- The cyst is unilocular and produces a well-defined radiolucency with sclerotic borders.
- The mandibular canal may be depressed by the cyst.

Treatment and Prognosis

The cyst is removed in-toto along with the impacted third molar and the surgical defect is thoroughly scrapped with a curette to remove all remnants. Recurrence is rare.

Inflammatory Lateral Periodontal Cyst (Lateral Radicular Cyst)

These are cysts arising from epithelial rests of Malassez due to inflammation of the periodontal ligament in the region. The inflammation of the ligament is brought about by pulpal irritation or pulp necrosis transmitted through a lateral canal or through an artificial perforation produced during an attempted root canal treatment procedure. This cyst is to be differentiated from the developmental lateral periodontal cyst, which arises in relation to a vital tooth.

The lateral radicular cyst is similar to the apical periodontal cyst in all aspects and therefore, needs no further explanation.

The Residual Cyst

A residual cyst is a cyst left behind in the bone of the jaws after the associated tooth has been removed. Although it is possible that this can occur with a variety of different types of cysts of odontogenic origin, in reality the persistence of a radicular cyst after tooth removal is the only firm evidence. These cysts predominantly are encountered in the mandibular premolar region.

The cyst is usually noted in an edentulous area and majority of the cases are middle aged or elderly. The cyst also tends to decrease in size as it ages and total resolution of residual cysts with intact linings has known to occur after a period of several years.

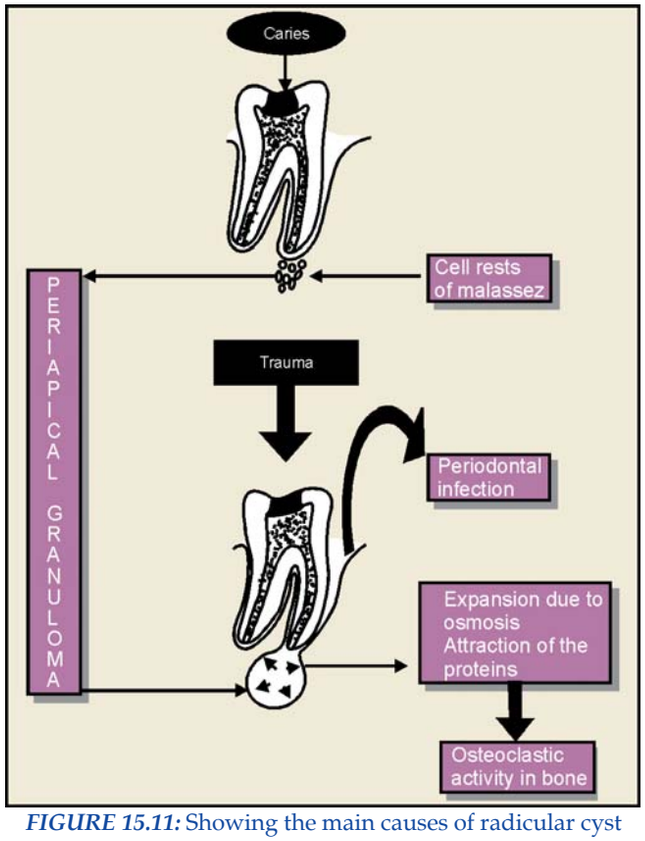


FIGURE 15.11: Showing the main causes of radicular cyst

The term "Residual Cyst", is rather loosely and inappropriately applied to:

1. A small cyst developing upon either a deciduous tooth or a retained root, which later exfoliates or is extracted without knowledge of the underlying pathology.
2. An undiagnosed developmental periodontal cyst after extraction of the permanent tooth.
3. The tooth associated with a lateral dentigerous cyst is removed but the existence of the cyst is unrecognized so that it persists and increases in size.

THE DEVELOPMENTAL CYSTS

The Odontogenic Keratocysts

Introduction

This cyst occupies a very special place amongst all the odontogenic cysts for the following reasons:

- It is the only odontogenic cyst that has very well defined histologic parameters that are constant in all cases and

is therefore the only odontogenic cyst that could be diagnosed solely by its histologic appearance.

- The pattern of enlargement and growth of this cyst is unique to it.
- This cyst offers the greatest challenge to surgical removal.
- This cyst has the highest rate of recurrence.
- This cyst is the most aggressive of all the odontogenic cysts.

The term ***Odontogenic keratocyst*** was coined by Philipsen in 1956. The term ***Primordial cyst*** has been unfortunately used synonymously with the odontogenic keratocyst. This term is applicable to those cysts that arise due to degeneration of the enamel organ and thereby occurring in place of a tooth.

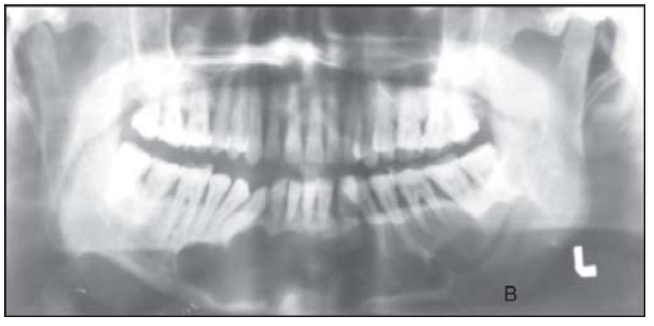
Not all odontogenic keratocysts are of this type, though primordial cyst may also arise in the presence of a full complement of teeth due to degeneration of the enamel organ of a supernumerary tooth. Not all primordial cysts present the required histologic criteria so very characteristic to an odontogenic keratocyst.

Primordial cysts in relation to mesiodens have been noted. These lesions are invariably small and innocuous, whereas the odontogenic keratocyst very rarely affects the anterior jaws and if it does so, the canine region is more commonly affected. The odontogenic keratocyst is usually a multilocular, destructive lesion of the jaws.

Pathogenesis

The epithelium of the keratocyst is strongly believed to arise from either the dental lamina or the residue of the dental lamina (cell rests of Serre). This organ of the tooth germ remains most active in the posterior jaws over a period when at other sites it has undergone involution.

Origin from the residues of the dental lamina remains the most likely situation. The dental lamina differs from the oral epithelium in that it has received the inductive influence of the appropriate underlying ectomesenchyme, although its differentiation has progressed only to an early stage of tooth development prior to histomorphogenesis of the enamel organ. The mesenchyme not only induces epithelial differentiation but also maintains these changes and different molecular species may be involved in two processes. Such mesenchymal influences may be crucial



FIGURES 15.12A to C: Shows clinical and radiographic pictures of patient with odontogenic kertaocyst.(A) extra oral draining sinus in the submandibular area. (B) OPG a large unilocular scalloped radiolucency with well defined margins extending from the molar area on the left side to the canine on the right with displacement of the roots. (C) Lateral oblique view (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

to the subsequent fate of these cells in cyst formation, because reciprocal induction of mesenchyme in developing cyst may be necessary.

Clinical Features

The cyst is most commonly encountered in the second and third decade of life.

The mandibular jaw is most commonly affected. In the mandible, the sites of preponderance are the third molar, ramus followed by the second and first molar area and then the incisor area in decreasing order.

In the maxilla, the third molar area is the most commonly affected followed by the canine region in decreasing order.

The age and site proclivity of the odontogenic keratocyst is similar to that of the ameloblastoma.

In early stages, the patient is asymptomatic and may approach for treatment due to pain, swelling and discharge. In such instances, the cyst is almost invariably secondarily infected.

- As the cyst expands, it tends to first involve the cancellous bone and only in the later stages is cortical plate expansion noted.
- The cyst may enlarge and involve almost the entire ramus, angle area as well as the body of the mandible before it produces a noticeables swelling causing facial asymmetry.
- The teeth in the region of the cyst may show mild to moderate degree of mobility due to loss of the supporting bone. The teeth are all vital unless otherwise afflicted by a pulpal pathology.

Radiographic Features

- The cyst usually presents as a multilocular radiolucency and shows greater involvement of bone than could be appreciated clinically.
- The radiolucency is well defined and the borders may appear sclerotic.
- Fine radiopaque septa traverse through the radiolucency giving it a *soap bubble* like appearance.
- The borders of the radiolucency extend between the roots of teeth and therefore appear scalloped.
- The radiographic evidence of root resorption is uncommon.
- The inferior dental canal may be pushed downwards and displaced.
- Large cysts with secondary infection may produce paresthesia in the lip region. (*Vincent's sign*)
- Some keratocysts may be associated with an impacted tooth and invariably this tooth is displaced as far as the coronoid or condylar process or to the lower border of the mandible, in which case, the cyst may be mistaken for a dentigerous cyst.
- The radiographic features of the keratocyst closely mimic those of the ameloblastoma, central giant cell granuloma and aneurysmal bone cyst (Figs 15.12 and 15.13).

FIGURE 15.13: A 42-year-old male presented with minimally expansile lesion of the mandible. Radiographically the lesion surprised the clinicians by its size and had horizontal trabeculations and edges non reactive. Biopsy revealed features of Odontogenic Keratocyst OKC (Iyengar A, Girish Rao, Nagesh KS RV Dental College, Bangalore 2004)

Histologic Features

The lumen: In most instances, the lumen contains keratin, which appears inspissated and dirty white resembling pus, but without the smell. This material is usually lost during tissue preparation. Otherwise, the lumen may contain a fluid whose total protein is below 4.0 gm/dl.

The epithelial lining: The epithelial lining in the keratocyst is highly characteristic and its features remain unchanged even in different specimens.

- The epithelial lining is composed of a layer some 6-10 cells thick. The lining maintains this uniform thickness with very minor changes.
- The surface of the lining is thrown into smooth folds giving it a "Corrugated" appearance.
- The transition of cells from the suprabasal to the keratinized is quite abrupt.

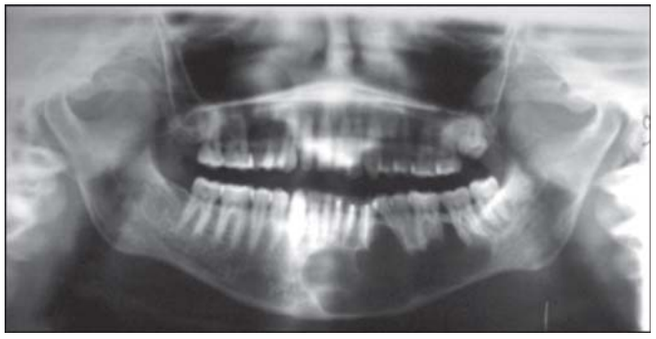


FIGURE 15.14: Benign tumors and cysts closely mimic each other. This was a 32-year-old male who came with mild discomfort in the left mandibular region of 3 years duration. OPG revealed huge radiolucent lesion with well demarcated reactive borders. The roots of adjacent teeth were resorbed. Histopathologically reported as Amelobalstoma (Sripathi Rao BH, Vidya Bhat, Bailoor DN 2004 Yenepoya Dental College, Mangalore)

The surface cells are usually parakeratinized, although orthokeratinization has now been recognized in these cysts. In such cases a rather prominent granular cell layer can be identified. The basal cells are tall columnar and possess an ovoid, darkly stained nucleus usually situated away from the basement membrane. This arrangement gives the basal layer a uniform appearance often described as a *Tomb Stone* like or a *Picket fence* like palisaded appearance.

- Metaplastic changes (Mucous cells, ciliated cells) are infrequent and so is hyaline body formation.
- The mitotic value of the suprabasal layer is very much greater than in epithelium of other cysts.
- The basement membrane region is flattened with no rete peg formation. Rete peg formation may be noted overlying areas of inflammation in the capsule.
- The epithelium is often noted to be detached from the capsule.

The capsule: It is characteristically thin and collagen fibers are less densely arranged when compared to other cysts.

- The capsule stroma is rich in glycosaminoglycans and proteoglycans.
- The capsule shows a scattering of fibroblasts and mast cells, though inflammatory cell infiltrate is relatively uncommon.
- Cholesterol crystals may be present within the capsule as in other cysts.
- Areas of mineralization may be seen.
- An important feature of the capsule of the keratocyst is the presence of islands of odontogenic epithelium that have formed daughter or "Satellite" cysts.

Treatment

- Careful enucleation: if site is accessible and lesion is small with regular follows up to detect recurrence, if any.
- Large multilocular lesions require enbloc resection followed by bone grafting.

Reasons for the High Rate of Recurrence

- Large lesions occupy relatively inaccessible sites.
- Nature of growth pattern with microscopic "burrowing" into bone, which is not identifiable radiographically or during surgical removal.
- Higher mitotic index of epithelial cells responsible for the relatively invasive nature of the cyst.
- Thin, friable capsule preventing complete removal. Capsule easily fragments and tears during removal. The nature of the capsule combined with an inaccessible site, may prevent its complete removal.
- Epithelial lining often and easily detached from the capsule. Part of it may fragment and be retained within bony cavity.
- Presence of satellite or daughter cysts within the bone wall.
- Active odontogenic epithelial islands within bone and adjoining mucosa.

Complications

- Recurrence of the cyst.
- Destruction of large parts of bone.
- Secondary infection and osteomyelitis with attendant features and complications.
- Pathologic fractures of the jaw.
- Development of ameloblastoma and carcinoma from cyst lining (Fig. 15.14).

Note: Multiple odontogenic keratocysts are associated with the Gorlin-Goltz syndrome.

The Primordial Cyst

This cyst develops through the disintegration of the enamel organ and its liquefaction before the formation of any enamel or dentin. This cyst, therefore, forms in place of a tooth rather than being associated with it.

The primordial cyst may arise due to cystic degeneration of the enamel organ of a supernumerary tooth and may hence be present with a full complement of teeth in the jaws. The synonymous usage of the term odontogenic keratocyst with the primordial cyst has led to much confusion and the authors believe that not all primordial cysts need be odontogenic keratocyst histologically.

Pathogenesis

The cyst arises as a result of liquefaction degeneration of the stellate reticulum of the enamel organ and the lining is probably derived from the cells of the outer and inner enamel epithelium.

Clinical Features

The cyst forms early in life but may be detected only later.

- The cyst is usually asymptomatic and may even be discovered accidentally, upon routine radiographic examination.
- The cyst expresses variable features and can thus even be large and destructive.
- It may be associated with a retained deciduous tooth. It appears in place of the underlying permanent tooth.
- The site of occurrence may vary from the anterior midline in the maxilla to the canine and the third molar and from the third molar area in the mandible to the premolar area.

Radiographic Features

The cyst appears as a unilocular radiolucency in place of a tooth.

- Borders of the radiolucency are well defined and may even appear sclerotic.
- The cyst may also be large and multiloculated with scalloped margins.

Histologic Features

The primordial cyst exhibits no specific histological features and may be indistinguishable from other odontogenic cysts.

Treatment

This contains enucleating the smaller lesions with thorough curettage of the defect. Larger lesions resembling the odontogenic keratocyst require treatment based on the guidelines of the keratocyst.

The Dentigerous Cyst (Follicular Cyst)

Introduction

The word 'Dentigerous' means "Tooth bearing".

The term was coined by Paget in 1863 for cysts of the jaws containing teeth. This cyst is the commonest developmental odontogenic cyst. It arises around the calcified crown of an impacted, embedded or unerupted tooth.

The cyst shows varying degrees of clinical involvement and may be unnoticed or may become aggressive and bone expanding.

Pathogenesis

The term follicular cyst would suggest an origin from the dental follicle that is a mesenchymal structure, which is highly unlikely.

- The lining of the cyst is attached to the cemento-enamel junction of the unerupted tooth. Therefore, it is obvious that the epithelial lining is derived from the reduced enamel epithelium covering the formed crown.
- The mechanism of fluid accumulation to form the cyst is unknown, most probably; mechanical disturbances during the eruption process may cause transudation or exudation of fluid from the follicular vessels into the space between the reduced enamel epithelium and the surface of the crown.
- Some degree of dilatation of the follicular space after crown completion and prior to eruption is usually noted.
- Suggestions have been made to explain an extra follicular origin of the epithelial lining from the residue

of the dental lamina. Such cysts would invariably be odontogenic keratocysts and would not assume a dentigerous relationship with the involved tooth.

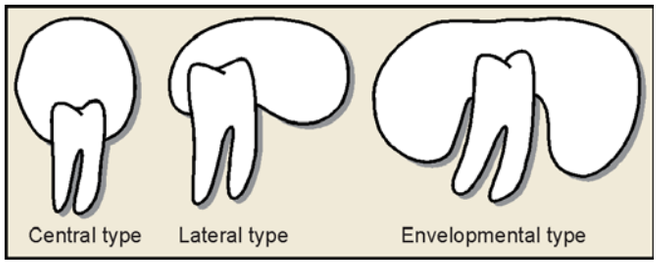
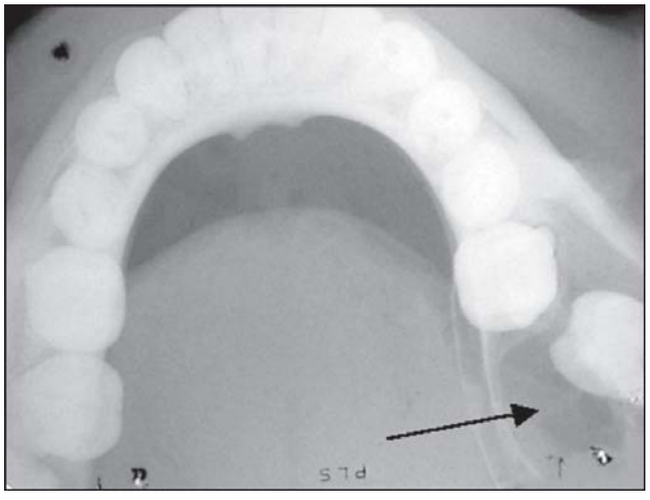


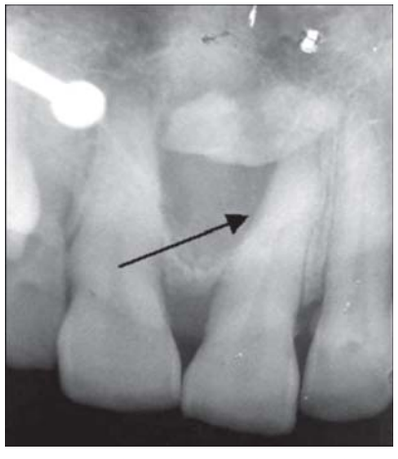
FIGURE 15.15: Some types of dentigerous cysts

Clinical Features

A tooth is usually missing from the arch, though these cysts could form around supernumerary teeth as well as around odontogenic hamartomas.

- The patient may otherwise be asymptomatic unless secondary infection supervenes accompanied by a sudden increase in swelling with pain and possibly a sinus tract formation with discharge.
- The cyst commonly occurs in the second or third decade of life when most of the teeth erupt into the oral cavity.
- The cyst is encountered in the region of the mandibular third molars followed by the maxillary third molars followed by the maxillary canine region, as these are the teeth most commonly prone to impaction.
- The cyst may show aggressive behavior with rapid bone destruction and expansion of the buccal and lingual cortical plates producing facial asymmetry.
- Though the cortical plates are expanded, the overlying mucosa appears normal and intact.
- Neighboring teeth may show mobility due to loss of supporting bone as well as due to some degree of root resorption.
- The cortical plates may be so thinned out that they can be easily compressed giving them a typical *Tennis ball* like consistency.
- As the cyst expands further, the cortical plates may be so eroded that they produce a *Egg shell crackling* when the two or more broken ends are moved against each other. This finding is reproducible as the broken ends regain their original positions due to elasticity of the bone accompanied by the hydrostatic pressure of the cyst.





FIGURES 15.16A and B: Show occlusal and IOPA radiographs of dentigerous cyst. The cystic radiolucency associated with unerupted tooth, causing expansion of the cortical plates; there is no resorption or displacement of teeth. (Omal PM, Beena K, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

- The cyst may become continuous with the oral mucosa and feel fluctuant and may seek to discharge its contents.

Radiographic Features

The cyst appears as a unilocular dilatation of the follicular space around the crown of an impacted tooth (Fig. 15.15).

- The more aggressive lesions tend to produce a scalloped, multiloculated radiolucency with a soap bubble like appearance simulating an odontogenic keratocyst or even an ameloblastoma, but the sclerosed boundaries are not usually seen.

- The lesion may be extensive and the radiolucency may reach as far as the coronoid or the condylar process or the lower border of the mandible.
- The impacted tooth may be displaced to the lower border of the mandible or anywhere in the ramus. Cysts of the maxilla may extend deep into the antrum.
- The adjacent teeth show some degree of root resorption.
- The bone of the neurovascular channel is usually resorbed and the neurovascular bundle is displaced by the expanding cyst (Figs 15.16 to 15.20)

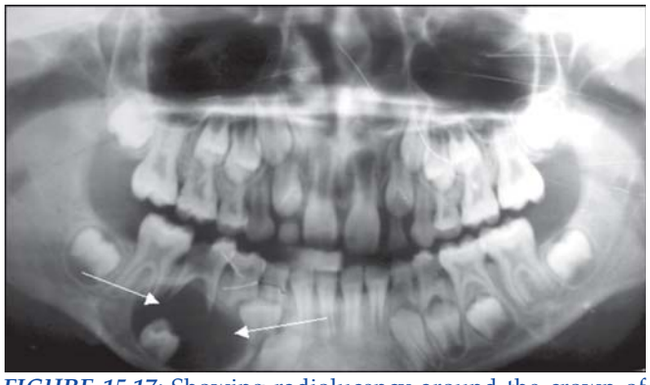


FIGURE 15.17: Showing radiolucency around the crown of unerupted second premolar. Is it a dentigerous cyst or a radicular cyst in relation to second deciduous molar? (Courtesy: Ani John, Hemant Umarji GDC Mumbai)

Histologic Features

The epithelial lining is of the stratified squamous type and usually shows no keratinization.

- The lining is thin with a regular layer of flattened or cuboidal cells, two to six cells thick.
- The basement membrane is flattened or may show very poorly formed rete pegs.
- The epithelial lining ends abruptly at the cementoenamel junction of the tooth.
- The squamous cells undergo mucous metaplasia quite commonly.
- Hyaline bodies or Russell's bodies are also occasionally seen.
- The epithelial lining may be orthokeratinized or may at times resemble the odonogenic keratocyst.
- The connective tissue capsule is usually thin, being composed of cellular fibrous tissue, containing few inflammatory cells.
- Inactive islands of odontogenic epithelium are commonly seen within the capsule.
- Cholesterol clefts, giant cells and areas of mineralization may also be identified within the capsule.

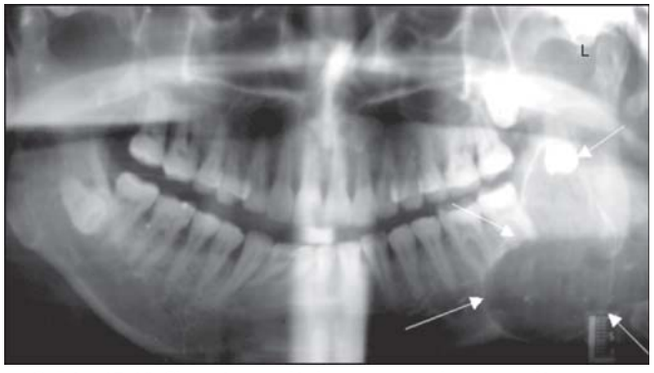


FIGURE 15.18: Showing radiolucency on the ascending ramus region left side surrounding an unerupted third molar tooth. Histopathology revealed Dentigerous cyst (Courtesy: Ani John, Hemant Umarji GDC Mumbai 2004)

FIGURE 15.19: Showing odontome together with a dentigerous cyst. This was surgically removed and confirmed histopathologically (Courtesy: Mani V et al, Calicut, Kerala. 2004)

Treatment

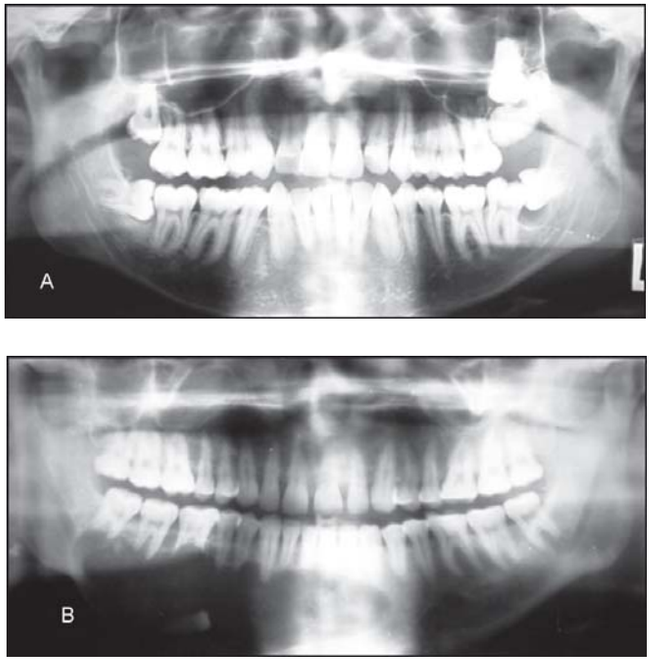
Small lesions could be enucleated and larger lesions are usually marsupialised to prevent fracture of the jaw and the defect fills by apposition of new bone from the periphery as the cyst membrane collapses.

Complications

Recurrence due to incomplete removal.

- Development of ameloblastoma from lining epithelium or rarely from odontogenic islands within the capsule.

- Development of squamous cell carcinoma from these epithelial sources.



FIGURES 15.20A and B: Depicting the dentigerous cyst with left mandibular horizontally impacted third molar. Size of the cyst is relatively small. See figure B where a massive dentigerous cyst can result in wanton destruction of the mandible and esthetic disfigurement (Iyengar A, Girish Rao, Nagesh KS RV Dental College, Bangalore 2004)

The Eruption Cyst

This developmental odontogenic cyst arises within the soft tissue around the alveolar ridge, overlying the crown of an erupting tooth. The cyst is associated with an interruption in the eruptive pathway of the tooth.

Pathogenesis

The crown of the involved tooth projects into the lumen of the cyst as is the case with the dentigerous cyst. Therefore, it may safely be assumed that the lining epithelium is derived from the reduced enamel epithelium.

In case of the dentigerous cyst, the disturbance in eruption occurs when the tooth is within the bony crypt, whereas in that of the eruption cyst, the disturbance occurs when the cyst is within the mucosal soft tissue.

Clinical Features

This cyst commonly occurs in the first and second decade of life.

- The cyst is associated with an erupting tooth and appears as a soft, fluctuant swelling over the alveolar ridge.
- The color may vary from normal mucosal pink to purple depending upon the type of fluid present within the cyst.
- When the fluid is blood tinged, the color is purple and the term *Eruption hematoma* is used.
- The cyst presents not much resistance to the erupting tooth. The cyst usually ruptures, discharges its contents into the oral cavity and permits the tooth to erupt normally.
- Otherwise, an incision is made to discharge the contents and thereby assisting in the eruption of the tooth (Fig. 15.21).

The Lateral Periodontal Cyst

This is a developmental odontogenic cyst that occurs infrequently and is seen arising from the periodontal membrane space of a vital tooth.

This terminology was used inappropriately in the past to include the lateral radicular cyst, the paradental cyst, the lateral dentigerous cyst and the gingival cyst of the adult.

Radiographic Features

The cyst usually presents as a unilocular radiolucency arising from the periodonal ligament space.

- The cyst boundary is well defined and often sclerotic.
- It does not cause root resorption.
- Some lateral periodontal cysts are multiloculated and are termed as "Botryoid odontogenic cysts". The word Botryoid means, like a bunch of grapes.

These multiloculated cysts may be mistaken as either a keratocyst or an ameloblastoma.

Treatment

The cyst is carefully enucleated with care to preserve the vital teeth.

Dental Lamina Cyst of the Newborn

Synonym

Gingival cyst of newborn.

Introduction

These are true developmental cysts of odontogenic origin. These were in the past misinterpreted as the "Predeciduous dentition". They have often been incorrectly called as "Epstein's pearls" and "Bohn's nodules".

Clinical Features

These cysts are present within the superficial aspect of the connective tissue overlying the alveolar ridge.

- The cysts are usually multiple but well separated from each other.

FIGURES 15.21A and B: A six month old child presented with a painless swelling which was fluctuant and bluish in color and translucency. No treatment was done and within a period of 20 days it ruptured on its own and the deciduous central incisor erupted normally. In rare cases the eruption cyst may have to be lacinated (Bailoor DN, Aruna N 2004 Yenepoya Dental College, Mangalore, Aruna is currently with DAV Dental College Yamuna Nagar, Haryana)

- They appear whitish and blanched.
- They cause no discomfort to the infant or the mother.

Treatment

These cysts require no treatment as they spontaneously rupture and discharge their contents into the oral cavity and their lining epithelium fuses with the oral epithelium.

Gingival Cysts of the Adult

Introduction

This cyst occurs infrequently.

Pathogenesis

The most acceptable hypothesis is that the lining is derived from the post functional cell, rests of the dental lamina as in the case with the lateral periodontal cyst.

Clinical Features

Usually seen in adults.

- The mandibular premolar and canine region are sites more commonly affected. The associated teeth are all vital
- The cyst appears as a small, well defined painless swelling on the gingiva. It may affect any part of the gingiva.
- The swelling is of the same color as the adjacent mucosa.

Treatment

Surgical excision of the cyst provides best results and the lesion does not tend to recur.

Calcifying Epithelial Odontogenic Cyst

Synonyms

Gorlin's cyst, Keratinizing and/or calcifying epithelial odontogenic cyst.

Introduction

This is a unique odontogenic lesion, which led many a pathologist to term it as a variant of an ameloblastoma or an odontoma, until Gorlin and his co-workers characterized it and coined the term in 1962.

Pathogenesis

The origin of this lesion is most uncertain though Gorlin and colleagues have proposed its origin from the enamel organ itself.

Various attempts have made to classify this hitherto unknown lesion. This is due to the variable natural history and histopathology of the lesion.

Praetorious and colleagues[1] have classified the lesion into the following groups:

Type IA: Simple unicystic type.

Type IB: Odontome producing type.

Type IC: Ameloblastomatous proliferating type.

Type II: Neoplastic type (Dentinogenic ghost cell tumor).

Clinical Features

This cyst presents no particular age or sex incidence.

- Though there is no preferred site in the jaws, majority of cases appear anterior to the first permanent molar.
- This lesion is known to arise within the soft tissue also.
- The lesion is usually asymptomatic and the patient notices only a slow growing swelling.

Radiographic Features

The lesion may appear as either unilocular or multiolcular radiolucency (Fig. 15.22).

- The margins may or may not be well defined.
- The lesion may be associated with an impacted tooth or it may show plenty of Denticle like structures.
- Spotted radiopacities characteristic of calcifications may also be observed.
- The lesion may easily be misdiagnosed as either an Adenoameloblastoma or a Pindborg's tumor.

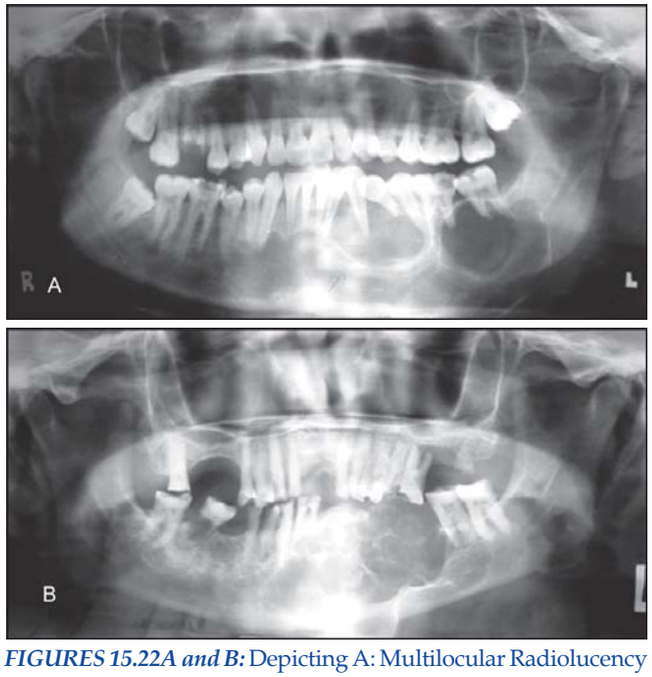
Histologic Features

These are quite variable and range from a simple cystic type to a solid tumor like mass resembling an ameloblastoma.

- Stellate reticulum like cells are seen above a darkly stained basal layer of cells that resemble ameloblasts.
- Many epithelial cells enlarge and undergo cytoplasmic keratinization and stain eosinophilic. These cells are called "Ghost cells".
- Many such cells would aggregate and form large keratin filled masses.
- Calcified masses may appear irregular and dystrophic although dentin like tissue is often identifiable due to the tubular pattern.
- Odontome like structures may also be formed by the lesion.

Treatment

Simple cystic type of lesions is usually enucleated while the more sold types would require wider surgical intervention.



FIGURES 15.22A and B: Depicting A: Multilocular Radiolucency with mild buccal plate expansion. Enucleation revealed radicular cysts of the mandible. B: Suspected to be cystic lesion initially, histopathologically reported as giant cell lesion. Serum chemistry was however normal. The delicate trabeculae in the lesion usually give a hint of the deviation from cystic pathology. (Courtesy of Iyengar A, Girish Rao, Nagesh KS RV Dental college, Bangalore 2004)

Nonodontogenic Cysts

These are true cysts whose epithelial lining is derived from sources other than those responsible for the formation of a tooth. The Retention type of cysts is commonly associated with secretory apparatus whose ducts have been obstructed. The Fissural or inclusion types of cysts are as

the name suggests, cysts that occur along lines of fusion of embryonic processes in the head and neck. These cysts arise from epithelial cells entrapped or included within fissures created during embryogenesis.

THE RETENTION CYSTS

Mucous Retention Cysts

Synonym

Mucous retention phenomenon, Mucocele.

Pathogenesis

The cyst arises due to dilatation of the glandular duct caused by the obstruction of free flow of its secretions. This obstruction is stated to be partial and could be in the form of an intraductal calculus or narrowing of the lumen of the duct due to a neighboring connective tissue scar contracture.

Clinical Features

This cyst occurs in the older individuals as compared to the extravasation type, which occurs in the younger age group.

- The cyst may form in almost any area of the oral cavity.
- The lesion may lie deep in the connective tissue and produce a slight elevation of the mucosa or it may be superficial and appear like a vesicle.
- Large retention cysts of the salivary glands occur commonly in the floor of the mouth in association with the ducts of the submandibular or sublingual glands.
- These slow growing, large, painless masses seen unilaterally in the floor of the mouth tend to elevate the tongue.
- These masses resemble the belly of a frog and are therefore, termed as "Ranulas".
- Large deep seated Ranulas may herniate between muscle fibers and reach into the neck or the superior mediastinum. Such lesions are called "Plunging ranulas".

Treatment

The retention cyst could be conservatively managed by removal of the superficial part of the lesion and large lesions may be excised with the involved gland.

Sebaceous Cyst

Synonym

Epidermal cyst, Pilar cyst, Steatoma, Wen.

Introduction

This cyst results from the obstruction to the duct of a sebaceous gland. The sebaceous glands produce their secretions by fatty degeneration of their central cells. These glands are therefore called as Holocrine glands. The duct of the gland opens most commonly into the hair follicle and at times on to the skin surface directly.

Clinical Features

The sebaceous cyst may occur anywhere on the skin surface but the more common sites are the scalp, the skin of the face and the scrotum, and seen in adult males.

- They usually occur as solitary swellings.
- The swelling ranges in size from a few millimeters to a few centimeters.
- The swelling is well demarcated, dome shaped and umblicated by the formation of a dark spot termed as the PUNCTUM.
- The punctum represents the point of blockage of the duct where the skin appears fixed.
- The cyst is soft in consistency and appears fluctuant. It is freely movable over the underlying tissue.
- On squeezing the cyst, a putty like cheesy material exudes from the puntum. This material consists of fat and degenerating epithelial cells.

Complications

- Formation of a sebaceous horn.
- Calcifications within the cyst.
- Rarely, malignant transformation into a sebaceous carcinoma.
- Infection and ulceration of the cyst.

FISSURAL (INCLUSION) CYSTS

The Globulomaxillary Cyst

Pathogenesis

There are various hypotheses attempting to explain the origin of this cyst.

- It is said to arise due to a proliferation of epithelial cells entrapped between the globular portion of the medial nasal process and the maxillary process.
- Due to a proliferation of epithelial cells situated in the suture between the premaxillary and maxillary bones.
- It is of developmental odontogenic origin.

Clinical Features

The cyst is seen between the lateral incisor and the canine of the permanent dentition.

- Bilaterally placed cysts have been reported.
- The cysts are usually asymptomatic and may be discovered accidentally.
- Swelling and pain is usually associated with secondary infection.
- The associated teeth are vital.

Radiographic Features

The cyst is placed between the roots of the lateral incisor and the canine.

- It splays the roots of these teeth away from each other and the crowns, therefore, appear to crowd.
- The cyst is an inverted pear shaped radiolucency whose borders are well defined but may not be sclerotic.
- At times the involved lateral incisor would demonstrate a deep lingual pit or would, in fact, be a dens in dente.

The Nasopalatine Duct Cyst

Synonyms

Incisive canal cyst, Median anterior maxillary cyst.

Introduction

The nasopalatine duct cyst is seen occurring deep in the bone. The more superficially occurring cysts in relation to the incisive foramen present as soft tissue swellings in the region of the incisive papilla. Such lesions are termed as the "Cysts of the palatine papilla".

Pathogenesis

This true non-odontogenic cyst is thought to develop from proliferating, traped, epithelial cells in the region of the nasopalatine duct.

Clinical Features

The cyst may occur at any age and some cases have been reported even in infants and children.

- If superficially placed, the lesion appears as a small, blue submucous swelling.
- Deeper cysts appear as small swellings in the anterior midline which may be compressible.
- The cysts may otherwise be symptomless.
- The cysts expanded slowly and their size remains static for long periods.
- The cyst may discharge a watery secretion at times.

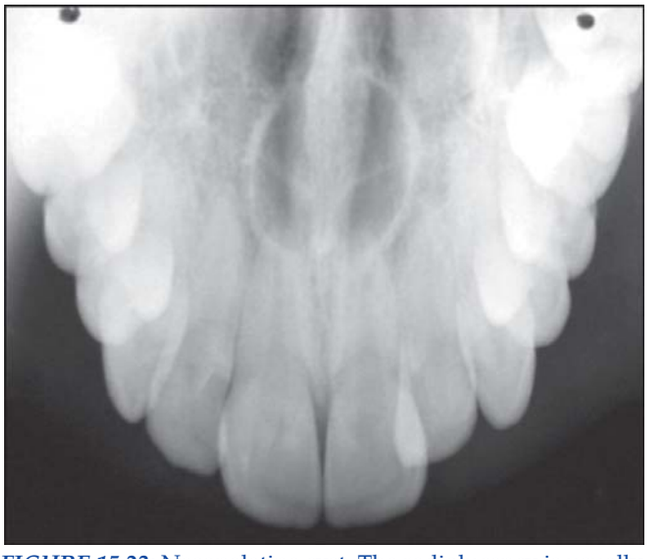


FIGURE 15.23: Nasopalatine cyst. The radiolucency is usually beautifully symmetrical and location and vital teeth usual give a strong indication to the presence of Nasopalatine cyst (Girish Rao, Iyengar Asha, Nagesh KS, RV Dental College, Bangalore 2004)

Radiographic Features

The cysts appear in the anterior midline as heart shaped unilocular radiolucencies that are symmetrically distributed around the midline.

The radiolucency is traversed in the midline by the radiopaque anterior nasal spine and the septal crest of the premaxilla (Fig. 15.23).

Median Palatal Cyst

This cyst arises within the maxillary jaw at the midline in the region of the fusion of the two lateral palatal processes,

usually in the region between the two maxillary canines or more posteriorly.

The lesion appears as a unilocular, well-circumscribed radiolucency in the midline, usually located in the region of the maxillary canines, posterior to the incisive canal.

Median Mandibular Cyst

This so-called fissural cyst of the mandible is a rare entity.

The radiograph reveals a well-demarcated radiolucency in the lower anterior region which may extend upto the premolars as well. The cyst may resemble a lateral periodontal cyst or a primordial cyst.

SOFT TISSUE CYSTS

Epstein's Pearls and Bohn's Nodules

Epstein's pearls and Bohn's nodules are true epithelial lined cysts of non-odontogenic origin while the gingival cyst of the newborn is odontogenic in origin arising from the dental lamina. Epstein's pearls appear as raised nodules along the midpalatine raphe, which are upto 2 to 3 mm in size. They arise from epithelial cells entrapped in the palatal mucosa during embryogenesis.

Bohn's nodules appear similar to the Epstein's pearls but are usually spread all over the palate with greatest concentration in the region of the junction of the soft palate with the hard palate. They arise from entrapped epithelial cells involved in the formation of minor mucous glands.

Nasoalveolar Cyst

Synonyms

Nasolabial cyst, Klestadlts cyst, Nasal vestibule cyst, Gerber cyst.

This cyst is uncommon. The cyst forms in the soft tissue of the nasolabial fold. It has now been suggested that the cyst arises from the remnants of the nasolacrimal duct. The lesion more frequently occurs in middle-aged women. It appears as a diffuse swelling of the upper lip obliterating the nasolabial fold and raising the ala of the nose.

Thyroglossal Tract Cyst

The development of the thyroid gland is initiated around the fourth week of intrauterine development. The initiation occurs in the region between the first and second branchial arches that would form the tongue partially. The area is called the Foramen caecum. From here the rudimentary gland grows downwards to its ultimate position in the neck. The descending gland leaves a hollow tube from its origin, the thyroglossal duct. The cells of the tube usually disintegrate in the intrauterine life itself, though in some cases, they may proliferate later in life and form this cyst. The site of occurrence may be anywhere from the tongue to the floor of the mouth or in the neck, though the commonest site is in front of the hyoid bone.

The cyst occurs in young persons as a soft swelling of variable size, which may be asymptomatic, and usually rises when the tongue is thrust out.

The cyst is lined by stratified squamous epithelium, ciliated columnar epithelium, a mix of both or a transitional form. Occasional cases of carcinomas developing from these cysts have been reported.

Heterotopic Oral Gastrointestinal Cyst

This very rare and unusual lesion may occur anywhere in the oral cavity. Such lesions have been known to occur anywhere in the gastrointestinal tract as well.

They arise due to ectopic or heterotopic collections of gastric mucosa undergoing cystic transformation.

Children and young adults are more frequently affected. It is said that the lesion arises due to cystic transformation of epithelium entrapped within developing cervical lymph nodes. The epithelium is probably glandular in origin. The cyst is commonly seen in children and young adults and occurs in the neck, either near the angle of the mandible or anywhere along the anterior border of the Sternocleidomastoid muscle.

The lesion is slow growing, freely movable and usually asymptomatic.

Dermoid Cyst

This soft tissue cyst usually occurs in the floor of the mouth, although lesions have been seen in the submandibular as well as the sublingual region.

The cyst may be present at birth or manifest in young adults. Cysts in the floor of the mouth elevate the tongue and cause difficulty in speech and mastication, whereas

cysts below the geniohyoid muscle produce a submental swelling.

The cyst is said to arise due to incorporation of embryonic germinal epithelium in between fusing mandibular and hyoid branchial arches. The cyst may be soft and fluctuant or firm in consistency.

PSEUDOCYSTS (FALSE CYSTS)

Aneurysmal Bone Cyst

This lesion is a distinct clinical entity described by Jaffe and Lichtenstein in 1942, which until then was reported as an atypical benign giant cell lesion of the bones.

Etiopathogenesis

Many researchers have hypothesized upon the origin and mode of formation of this unique lesion.

- Locally altered hemodynamics causing increased venous pressure and engorgement leading to bone resorption and formation of a cystic area.
- Attempt to repair or resolve an intraosseous hematoma.
- Degeneration of other primary intraosseous lesions like giant cell granulomas or hemangiomas.

Clinical Features

The lesion is seen mostly in children and young adults.

- In the jaws, the mandible is more commonly affected and the lesion usually occurs in the body, angle or ramus area.
- Some patients may present with a past history of trauma at the affected site.
- Cases may be asymptomatic.
- The lesion expands the jaws and subperiosteal bone deposition may produce a hard swelling.
- The lesion may also perforate the cortex producing soreness and limitation of ability to move the bone. w During surgical intervention, the lesion appears like a "Blood soaked sponge".[10]

Radiographic Features

The lesion appears as an unilocular or "Soap bubble" like appearance.

The lesion expands the cortical plates and appears to eccentrically balloon or blow out the bone.

- Subperiosteal bone deposition may be seen as an intact thin shell.

Static Bone Cyst

Synonyms

Developmental bone cyst, Latent bone cyst, Stafne's bone cyst, and Lingual mandibular salivary gland depression.

This unusual lesion is in reality to be considered as a developmental defect rather than pathology.

The so-called cyst is actually a smooth well-circumscribed saucer shaped depression on the lingual surface of the body of the mandible. Such depressions are also recorded in the digastric fossa and sublingual region of the mandible. These depressions correspond to lobes of the submandibular or the sublingual salivary glands that occupy such concavities in the bone. In rare instances, the glandular tissue may actually be entrapped within the bone. The lesion is seen as a well-circumscribed radiolucency, which is round to oval and lies usually below the mandibular canal.

Traumatic Bone Cyst

Synonyms

Hemorrhagic bone cyst, Solitary bone cyst, Idiopathic bone cavity.

This cyst is a solitary lesion in the jawbones.

Pathogenesis

The following are probable factors put forth to explain the etiopathogenesis. Trauma causes intramedullary hemorrhage with bone resorption. The relatively high frequency of trauma to the jaws and paucity of such lesions disproves this hypothesis.

- Localised alteration in the calcium metabolism.
- Degenerating intraosseous tumors.
- Low-grade infection.
- Developmental aberration.
- Ischemic necrosis of the medullary space.
- Abnormal vascular channels and obstructed lymphatic flow.

Clinical Features

The cyst is usually noted in children.

The mandible is more commonly affected and most cases appear in the molar region.

- Lesion may be asymptomatic and discovered only during routine radiography.
- Teeth in the involved area are vital unless otherwise affected by pulpal necrosis.
- The lesion upon opening may be empty or contain little fluid and blood clots.
- Larger lesions may have eroded the mandibular canal and the neurovascular bundle may be easily identified.

Radiographic Features

The smaller cysts appear as unilocular radiolucencies usually above the mandibular canal in relation with the roots of the associated teeth.

The lamina dura around the roots may be seen to be intact upto some distance towards the apex after which it fades gradually.

- The cyst may closely resemble a radicular cyst and the vitality of the standing teeth should be assessed to prevent unnecessary sacrifice of these teeth.
- The cyst may be large, covering a wide portion of the mandible and may displace the mandibular canal towards the lower border of the mandible.
- The margins of the cyst extend between roots of teeth and appear scalloped, though sclerosis is uncommon.
- The roots may be splayed apart but realign after treatment.

Treatment

It is said that an exploratory biopsy that causes intralesional bleeding, may instigate and hasten healing. A curettage with placement of bone chips to fill the defect causes remission.

Mucous Extravasation Cyst

This is a type of mucocele that occurs as a result of pooling of mucus within the connective tissue of the mucous membrane as a result of severance of the duct that discharges the secretions into the oral cavity.

This cyst occurs commonly in young adults. The mucosa of the lip is commonly affected.

CONCLUSION

Cysts are slow growing lesions and usually do not cause a life-threatening emergency. But their early diagnosis and proper surgical treatment is necessary to prevent the esthetic damage and in some cases like odontogenic Keratocyst frequent recurrences requiring expensive hospitalization.

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Reddi Ramachandra

16 Oral Precancer

INTRODUCTION

The concept of oral precancer is especially relevant to the Indian subcontinent.

- Because of cultural habits of Betel nut, Betel leaf chewing, reverse smoking.
- Differing faiths and religious belief, which may make the food selection lopsided
- The assault on the oral mucus starts from a very young age due to highly spicy food habits.
- Genetic structure of Indians itself may predispose to the increased incidence of conditions like oral Submucous fibrosis?

  Oral Precancer then is an intermediate clinical state with increased cancer risk, which can be recognized and treated, obviously with a much better prognosis compared to the full blown malignancy.

  Precancer is divided into precancerous condition and precancerous lesions for sake of didactics and understanding.
- Oral Precancerous Condition is defined as generalized alteration in the state of condition of Oral mucosa associated with a significantly increased risk of malignant transformation.
  - Oral Submucous Fibrosis (OSMF)
  - Sideropenic dysphagia
  - Syphilitic Glossitis
  - Xeroderma pigmentosum
- Oral Precancerous Lesion is defined as a localized area of morphologically altered tissue where cancer is more likely than its normal counterpart.
  - Leukoplakia
  - Erythroplakia
  - Erosive Lichen planus
  - Bowen's disease
  - Dyskeratoses congenita
  - DLE—Discoid Lupus Erythematosus
  - Stomatitis Nicotina

DIAGNOSTIC STEPS

The private practitioner can do one of the following tests to confirm his clinical suspicion-

a. Toludine Blue staining
b. Exfoliative cytology
c. Biopsy: Incisional and excisional
d. FNAC—Fine needle aspiration cytology
e. OraScan Dx Brush Biopsy Technique using computerized diagnostics
f. Use of Vizilite®

Gold standard still remains the incisional biopsy sent to the histopathology laboratory in 10% formalin.

PRECANCEROUS CONDITIONS

Oral Submucus Fibrosis (OSMF)

In 1952, a condition called as 'Atrophia Idiopathica mucosae oris' was described for the first time by Schwartz.[1] Through the last three decades a gradual evolution of terms has resulted in a widely accepted name for this disease—Oral Submucous Fibrosis or OSMF.[9-11]

In South India it is characterized by severe burning sensation, inability to open the mouth, tongue rigidity, pallor and generalized stiffness of the mucosal tissues. This is explained by the autoimmune fibroelastic change-taking place just below the epithelial surface. Pharyngeal and esophageal changes have also been noted in this condition. It is a premalignant condition observed in the four states of South India commonly.

The Incidence rates of OSMF are available from India (Gupta et al 1980[2]). In Ernakulum in south, Incidence was reported as 8 per 100,000 Men and 19 per 100,000 Women per year. A study from Mangalore shows the malignant transformation of 2 out of 12 cases being followed up for a period of more than 3 and half years. Bailoor DN[3]1993.

The clinical features in this region are initially only burning sensation and pale colored taughtness spreading slowly from buccal mucosa, posterior tonsillar fauces. The burning becomes more and more pronounced and inability to open the mouth, and tongue rigidity becomes pronounced. In the latter stages only one finger opening remains and large erythematous patches and irregular ulcers set in. The vesicular lesions classically mentioned by some authors (Pindborg et al[4]) are not seen in the South Kanara and North Kerala populations (Fig. 16.2).

Review of Literature

OSMF is found to coexist in mouths of 40 percent of the oral cancer patients and was considered as precancerous. In a long term follow up study 7.6% of a series of 66 patients of OSMF underwent malignant transformation Murthy and Bhonsale.[5]

Two other studies, however, gave much lower figures (see Table 16.1).

Merchant A et al (2000)[31] have investigated the role of "Paan" in the development of the precancer conditions. "Paan" is a quid of piper betel leaf that contains areca nut, lime, condiment, sweeteners, and sometimes tobacco. They did this study to clarify the independent association of paan and oral cancer. People with OSMF (oral submucous fibrosis) were 19.1 times more likely to develop oral cancer than those without it. People using paan without tobacco were 9.9 times, more likely to develop oral cancer as compared with non-users, after adjustment for other covariantes.

Table 16.1: Malignant transformation of OSF in long-term Indian studies

Author	*Year*	*% transformation*	*Duration of study*
Gupta et al[2]	1980	2.3	10 years
Pindborg et al[4]	1984	4.5	15 years
Murthy and Bhonsale[5]	1985	7.6	17 years

Risk Factors

Major risk factors in our clinics appear to be persistent use of powdered areca nut (flavored suparis) all over India.

- Traditional habit of using Betel leaf, areca nut and lime continuously over long periods of time.
- Nutritional deficiencies due to food fads like vegetarianism or poverty. B complex and Iron Deficiency appear to strongly predispose the oral mucosa to the sub-epithelial and juxta-epithelial reaction followed by the fibro-elastic change of the lamina propria with subsequent epithelial atrophy. Long term ingestion of spicy food commonly seen in the south Indian states may be a contributing factor[19,20] (Fig. 16.1).

Diagnosis

Diagnosis is based on

- Clinically discernible blanching and pallor.
- Palpable bands
- Restriction of mouth opening.
- Severe burning sensation of mouth, aggrevated by use of even moderate spicy food.

Biopsy report characteristically showing histopathologically.

- Atrophic oral epithelium
- Loss of rete pegs
- Epithelial atypia may be observed
- Hyalanization of collagen bundles
- Fibroblasts decreased and blood vessels obliterated.

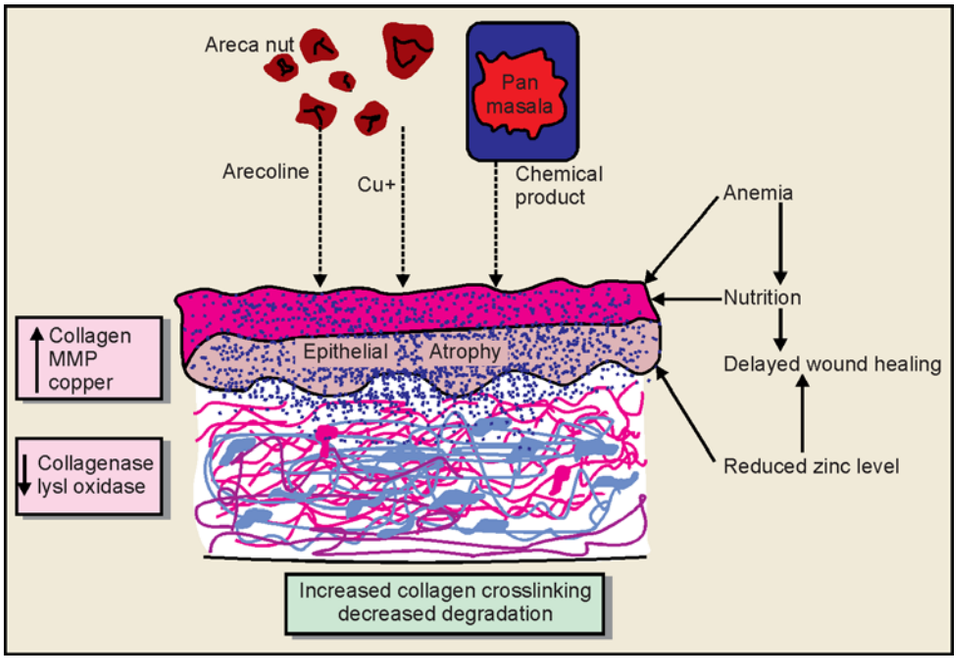


FIGURE 16.1: Proposed pathogenesis of oral submucous fibrosis

When the condition is diagnosed in our departments, we follow the staging given below (Some values taken from Mathur and Jha (1993).

Staging

Stage 1: Early OSMF (Fig. 16.3)

- Mild blanching
- No restriction in mouth opening
  - Normal - Distance between Central incisor tips
    - Males 35-45mm
    - Females 30-42mm
- No restriction in tongue protrusion
  - Normal -Mesio incisal angle of upper central incisor to the tip of the tongue when maximally extended with mouth wide open
    - Males 5-6 cms
    - Females 4.5-5.5 cms.
- Cheek flexibility…
  - CF = V1-V2

    Two points measured between;

    V_2 = is marked at one third the distance from the angle of the mouth on a line joining the tragus of the ear and the angle of the mouth,

    V_1 = the subject is then asked to blow his cheeks fully and the distance measured between the two points marked on the cheek

    Mean value for males…1.2 cms, Females…1.08 cms
- Burning sensation only on taking spicy food, or hot temperature liquids, etc.

Stage 2: Moderate OSMF…(Fig. 16.4)

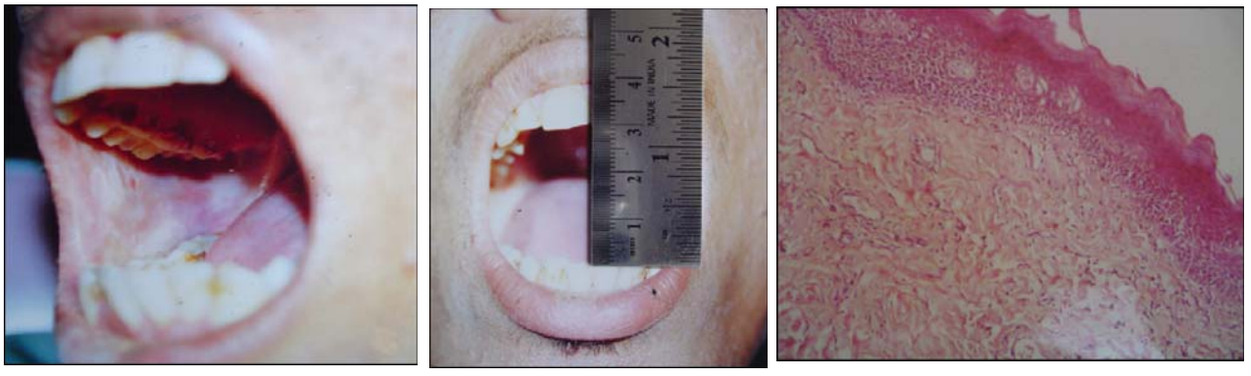
- Moderate to severe blanching.
- Mouth opening reduced by 33%.
- Cheek flexibility also demonstrably decreased.
- Burning sensation even in absence of stimuli.
- Palpable bands felt.
- Lymphadenopathy either unilateral or bilateral.
- Demonstrable Anemia on hematological examination.

Stage 3: Severe OSMF…(Fig. 16.5)

- Burning sensation is very severe. Patient unable to do day to day work
- More than 66% reduction in the mouth opening, cheek flexibility and tongue protrusion
- The tongue may appear fixed.
- Ulcerative lesions may appear on the cheek
- Thick palpable bands.
- Lymphadenopathy bilaterally evident.

FIGURES 16.2A to D: Showing clinical and histopathologic pictures of a patient with OSF grade II different areas of the oral cavity. The buccal and labial mucosa shows blanching and whitish discoloration. The histopathology shows epithelial atrophy and juxtaepithelial hyalinization and inflammatory infiltrate (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

***FIGURES 16.3A to C*:** Stage 1



FIGURES 16.4A to C: Stage 2

FIGURES 16.5A to C: Stage 3

Definite Nutritional compromise can be established in B complex (angular cheilitis) and Iron deficiency group.

Normally it is best to treat the OSMF in a hospital or Dental college set up, since the time and expertise required for management is available at these centers. The general dental practioners role in early diagnosis cannot but be overemphasized.

Treatment

Over all..

- Strict discontinuance of chewing habits both arecanut and betel leaf.
- Going on bland food, free from chillies and peppers..
- Nutritional support of high calcium and high protein combined with Iron supplementation.

  Capsule Raricap® Biostar®

  Capsule Fefol-Z® Oxyvit®

  Syrup Alprovit® tonic or Protone® granules + milk could be recommended.

- ***Stage 1:*** If the pallor reduces and burning sensation abates then keep under regular check up. Otherwise give the patient intralesional steroids 3 times a week by multiple puncture method for 6 weeks.
- ***Stage 2:*** It is managed by a mixture of
  - Intralesional injections of Hayluronidase + corticosteroids
  - Colloidal Iodine has been used with some success[18]
  - Placentrex an injection of placental extract intralesionally.
- ***Stage 3:***
  - Surgery with rotation flaps have been advocated
  - Systemic steroids complemented by local proteolytic enzymes such as Seradase® or Hyalase®.

Canniff et al[6] elucidates the interaction of genetic predisposition and Areca nut alkaloids in his hypothesis for etiopathogenesis.

The aetiological role of chillies gained support from the experimental observation of Sirsat and Khanolkar[7]

who elicited the submucous fibrosis type of reaction in Winstar rats by applying the extract of 'capsaicin' an active principle of chillies. The reaction was enhanced when the treatment was super imposed upon the dietary protein or vitamin deficiency.

Ramnathan's study[8] from Malaysia observed that 10 out of 13 OSMF patients had long-term Iron and Vitamin B complex deficiency and he hypothesized that this disease could be the Asian version of sideropenic dysphagia.

Treatment Regimens—used in different parts of India are briefly reviewed.

Sinha and Jain[12] have tried local injection of hydrocortisone 1.5cc for one group and 2.0cc of Placentrex once a week for the other group for 12 consequent weeks. They reported failure rate 7.2% for hydrocortisone and 31.3% for placentrex and concluded hydrocortisone was superior.

Kakar, Puri et al[13] recommended four different regimens.

- 4 mg dexamethasone biweekly
- 1500 iu of Hyaluronidase with 1 cc of Lignocaine biweekly
- 4 mg dexamethasone and 1500 iu of Hyaluronidase
- 2cc of Placental extract biweekly.

They concluded that no 3 combinations locally for seven weeks could give maximum improvement if it is followed by three weeks of Hyaluronidase.

Ramanjaneyalu and Prabhakar Rao[14] advocated 2 cc of placentrex injection intralesionally at weekly intervals for 10 weeks. They have found it was superior to cortisone. Two cortisone resistant cases also responded well to placentrex.

Gupta et al[15] reported that OSMF cases can be managed by Physiofibrolysis and that it could be of value in early and moderately advanced cases.

In Davangere the use of Levamisole together with Vitamin A 50,000 iu is being tried with considerable success by Balaji Rao.[16] This study stresses that other types of immunomodulation together with chemoprophylaxis may be tried.

A report from Hyderabad by Reddi R[17] suggests the use of Vitamin E concomitantly with the Hyalase + Betamethasone intralesional injection as compared with Hyalase and Betamethasone alone.

Gupta S et al (2004)[36] reported in their study that the decrease in beta-carotene and vitamin E was more significant in oral submucous fibrosis patients. After six weeks of oral administration of beta-carotene and vitamin E, patients showed increase in plasma level of these two antioxidants along with decrease in Malonaldehyde (lipid peroxidation product) level associated with clinical improvement.

The hypothesis of Vitamin E effect mechanism is…

- Preventing the oxidation of essential cellular constituents such as the formation of oxidation products.
- Act as scavengers of free radicals.
- Preventing the progression of Neurological abnormalities.
- Treatment with Vitamin E may improve the survival of Erythrocytes.

Bailoor DN, Prasanna K[37] (2004) in the study treated 18 OSF patients with intralesional combination of Betnesol® and Hyalase® (Once a week for 12 weeks) with Antioxidant (ALA 100® daily OD). They found significant reduction in burning sensation and improved mouth opening. This was more significant in antioxidant group compared to control group who were given only intralesional injections. They recommend the use of antioxidants at least for a period of three months initially for quicker results and nutritional support.

Gupta PC, Sinor PN et al (1998)[28] attributes the OSMF directly to the use of areca nut in various forms. Their study from Bhavnagar district found that people mostly used "mawa" a mixture of tobacco, lime and areca nut and that 10.9% of its users had OSMF. They confirm an increased prevalence of OSMF in lower age groups and have raised concerns that increase in the oral cancer incidence could be seen in the coming years.

Haider SM et al (2000)[30] have tried to stage the OSMF into clinical staging of A, B and C. **A** when the band is felt initially in the posterior region (interincisal opening > or = 20 mm) Type **B** when the bands are palpable in posterior strongly and buccal and labial initially (inter-incisal opening between 20 and 10 mm) and Type **C** when the bands are stongly felt in all the posterior, buccal and labial mucosa (inter-incisal opening of less than 10 mm). They have confirmed from their study of 288 patients from

Karachi (Pakistan) that the fibrous bands always developed first in the faucial regions and gradually involved the anterior aspects like buccal mucosa and the labial mucosa. This classification is also fairly simple and can be used by the dental practitioners.

Gupta PC (1999)[32] has compared the age specific incidence rates of mouth cancer (ICD 143-5) during 1983-87 and 1995 in the city of Ahmedabad shows that the incidence has significantly increased in the younger population (< 50 years). If this is seen in background of increasing prevalence of oral submucous fibrosis, especially in younger individuals, caused by gutkha, an industrially manufactured food item. It is concluded that urgent public health education measures are required to curb this new epidemic.

Sideropenic Dysphagia (Paterson-Kelly Syndrome; Plummer-Vinson Syndrome)

This syndrome is characterized by Iron deficiency anemia, dysphagia, Koilonychia (brittle, dry, flat and spoon-shaped), Glossitis, and Angular cheilitis.

In south India we observe more bald tongue cases rather than Glossitis. Glossodynia is a common finding, especially to spicy food. Middle aged and menopausal and postmenopausal women are seen to suffer from this condition. The dysphagia is caused due to the esophageal webs. The mechanism explained is that anemia causes epithelial atrophy, change in cell kinetics, and decreases the repair capacity of the mucosa. This allows the carcinogens and co carcinogens to act more aggressively, predisposing the entire oral cavity and esophageal area to malignancy.

In the clinics if any female patient is seen complaining of burning sensation, bald tongue and difficulty in swallowing of varying degrees then be sure to advice a complete hemogram, and a barium swallow. If frank ulcerative changes or advanced atrophic changes are seen immediate endoscopic biopsy, barium swallow and follow up is a must. Such cases are best treated in Hospitals in conjunction with Gastero-enterology specialists.

Syphilitic Glossitis

This effect on the tongue is seen in the late syphilis stage or the tertiary stage. The widespread use of antibiotics has considerably reduced the incidence of this stage in our clinics in India. It is characterized by atrophy of the filiform and fungiform papilla of tongue. This is hypothesized to be due to the obliterative endarteritis, which reduces the circulation to the superficial areas of the tongue. Mostly seen in the males and is regarded as a precancerous condition and any ulcerative or leukoplakia like lesions occurring in this area should be biopsied and treated aggressively in a STD clinic.

Xeroderma Pigmentosum

It is a genetically inherited skin condition in which there is a defect at the sub cellular level in the DNA repair mechanisms. Entire skin shows an abundance of pigmentation and skin gets easily afflicted with melanomas and squamous cell carcinomas.

THE PRECANCEROUS LESIONS

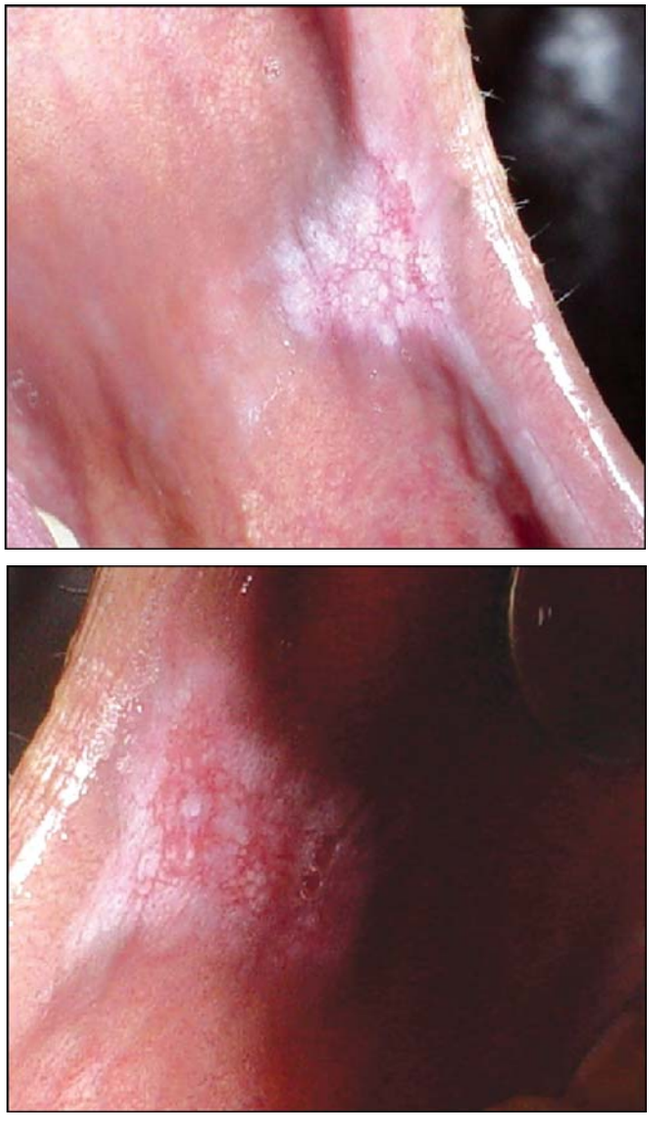
Leukoplakia

This lesion is discussed in detail in the White lesions chapter No 12. Important factor here is to evaluate the fact whether it is speckled or non speckled and check the site floor of the mouth vs rest of the mouth. All the floor of the mouth and speckled Leukoplakias need to be biopsied immediately in the dental clinic. All dental Surgeons must keep small bottles and supply of Formalin (10%) or absolute alcohol for fixing these biopsies, often a good incisional biopsy looses its diagnostic value because the dentist doesn't fix it properly and the histopathology technician cannot get a good slice!

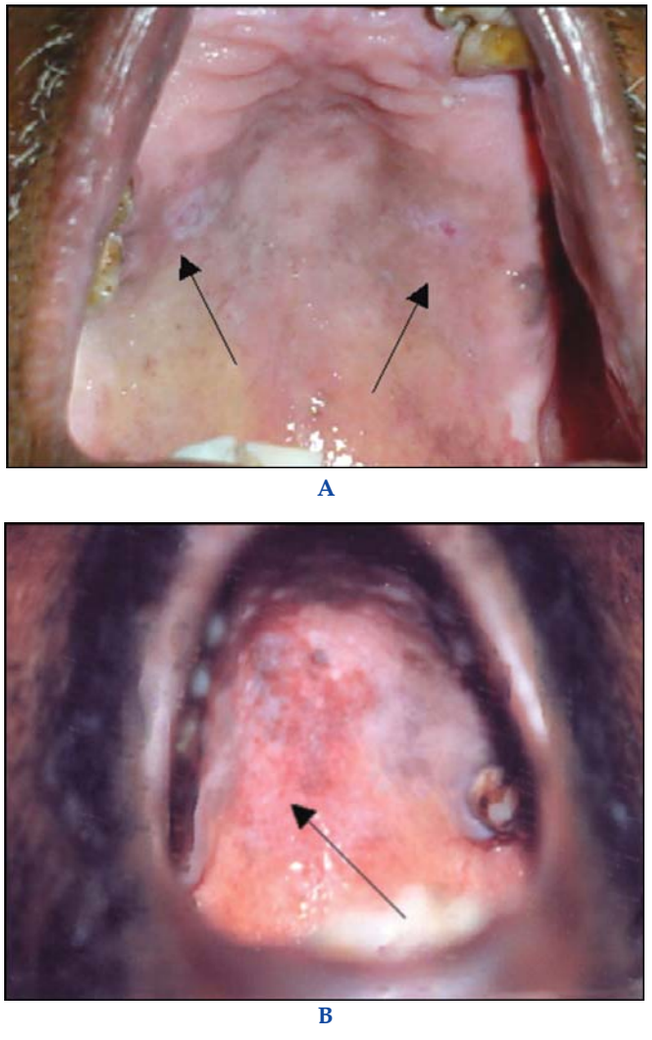
Gupta PC and Herbert JR et al[29] in their study from Ernakulam district in south India found that consumption of vegetables and fruits and several micronutrients notably Zinc in men and Iron in women resulted in significant protection in decreasing the risk of malignant conversions. Nutritional counseling on part of the dentist has become an important aspect of precancer care (see Figs 16.6 and 16.7).

Erythroplakia

This is defined as a persistent velvety red patch that cannot be identified as any other specific lesion and may be related to the tobacco abuse. The term erythroplakia should be seen as a potentially very serious lesion and immediate



FIGURES 16.6A and B: Shows clinical pictures of speckled leukoplakia. Reddish white discoloration with nodular white areas, which was non-scrapable and seen in a patient of 25 yrs with a habit of *bidi* smoking since 10 yrs (Omal PM, Beena K, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)



A

B

FIGURES 16.7A and B: (A) Whitish nonscrapable areas on the palate in a habitual bidi smoker. Indicative of leukoplakia. (B) Reddish white discoloration associated with burning in a 53 year old male who had habitual pan chewing and cigarette smoking since 35 years indicative of erythroplakia. (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

biopsy and later treatment procedures should be left to the nearest oncology center or major hospital.

Shafer and Waldron[21] have found that in their series 51 percent of the 58 cases to be invasive carcinoma. Floor of the mouth is the most commonest site for the males and gingival and alveolar mucosa is the most common for females.

Mashberg's and Morrissey's [22] recommendation that if any red lesion persist for more than 14 days after the removal of local causes and infection then immediate biopsy is mandatory. Histopathologically it is the same as Bowen's disease.

Stomatitis Nicotina

This lesion is directly attributed to tobacco abuse in form of cigarette smoking, *bidi* smoking or chutta smoking. In

areas of Andhra Pradesh where the reverse smoking habit is widely prevalent this condition is more common. Dental surgeon is likely to see this lesion in three stages.

Stage 1: White popular area with mild border of erythema, symptom less and accidentally discovered.

Stage II: The classical par-boiled appearance of mucosa interspersed with red dots. The red dot situated in center of the nodule represents an inflamed dilated orifice of the minor salivary gland. Up to this stage we observe that this lesion dramatically reverses on cessation of the smoking habit.

Stage III: Here the lesion is more than 1.5 cm in diameter and apart from the red dots interspersed ulcerated areas are evident. It this stage we consider the lesion as an *avatar* of speckled leukoplakia itself and hence with a very high pre-malignant potential.

In this stage all the steps mentioned for treatment of leukoplakia should be followed and referral to a teaching dental institution is a good idea!

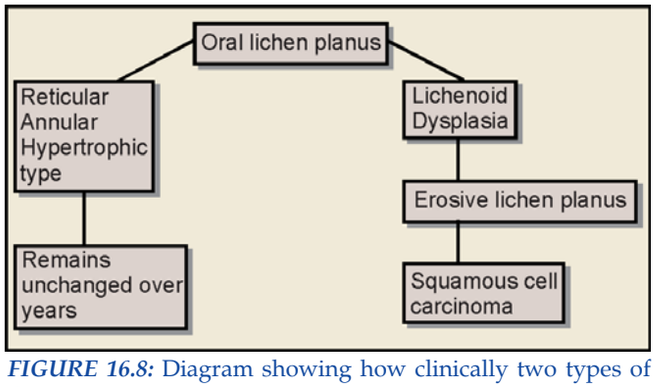


FIGURE 16.8: Diagram showing how clinically two types of OLP lesions may occur. The hypertrophic lesions usually go on for years, whereas the lesions that give rise to dysplastic changes early on, are termed lichenoid dysplasia which has higher risk of Ca conversion.

Erosive Lichen Planus

This form of Lichen planus is painful and the patient often comes with the chief complaint of pain or burning in that area. Clinical examination reveals an ulcerated or erosive red area with lacy *wickhams striae* at the edge of the lesion (Fig. 16.9). Here again we advocate immediate biopsy of the lesion, and local pain control with ointment Mucopain® or Lignocaine® and post biopsy infection can be controlled by oral penicillins like Oracyn K® and Pentids 200, 400 or 800®. Intra-lesional corticosteroids reduce the pain and reduce the pain and reduce the healing time. We have often used Amphotericin lozenges 10 mg about five to six times a day to control fungal component of this diseases seen in many patients. Sehgal[23] has reported the use of 500 mg of Griseofulvin per day for four weeks to be giving good relief for Lichen planus. Falk[24] and Beck[25] have reported some success with the use of Dapsone (Diamino diphenyl sulfone) over long time period of many months.

Balato[26] has used topical Cyclosporine for treatment with variable success. Since this disorder is still in the realm of unknown etiology, Conserative treatment in non-erosive and aggressive treatment in erosive varieties is recommended.

Campisi G[35] et al (2004) suggested that the new topical drug delivery system of lipid microspheres loaded with 0.025% of clobetasol propionate enhances symptomatic remission and compliance in OLP therapy.

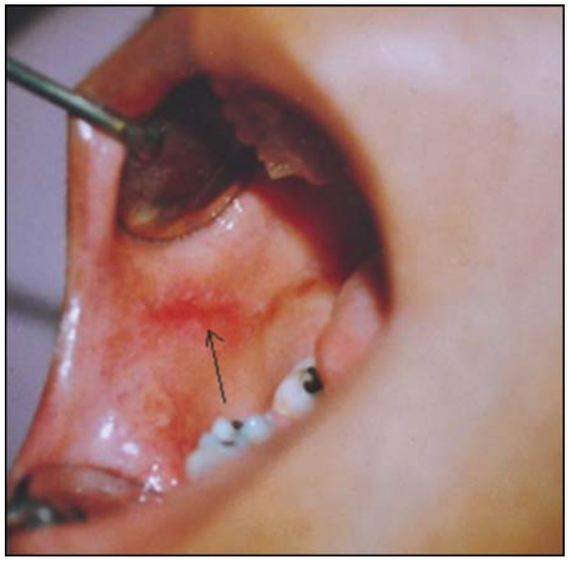
Recent literature is abounding with controversy regarding the malignant potential of lichenplanus.[33] According many authors those lesions that turn onto malignancy should be termed as lichenoid dysplasia.[34]

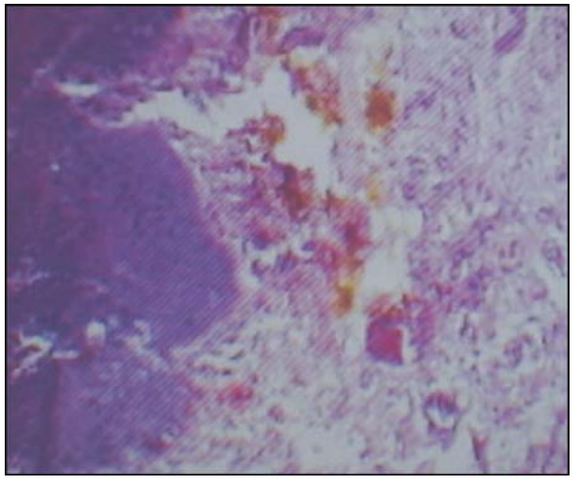
Lichenoid dysplasia resembles lichen planus in its clinical features, it may have a erosive component and be unilateral, exposure to carcinogens predispose it towards malignancy (Fig. 16.8). The histopathological features that distinguish it from lichen planus are (adapted from Eisenberg E):[33]

- Unusual to find basal cell liquefaction
- Lichenoid infiltrate is sparse
- Epithelial topography is suggestive of dysplasia
- Stratification disarray
- Maturational disturbance
- Nuclear enlargement
- Cellular pelomorphism
- Nuclear: cytoplasmic ratio reversal
- Dyskeratosis
- Individual cell necrosis
- Abnormal and increase in mitosis.

Bowen's Disease

This is characterized by scaly or plaque like lesions of the skin and is often seen in individuals who consume arsenic





FIGURES 16.9A and B: Showing 35-year-old female with an erosive lesion on the buccal mucosa, a clinical diagnosis of Erosive Lichen planus. Histopathology was report Lichenoid dysplasia (Nillofer S, Prasanna K, Bailoor DN, 2004, Yenepoya Dental College Hospital, Mangalore)

either by accident or as treatment in alternative medicine regimens. These red scaly areas enlarge into yellowish lesions which when removed leave granular surface with minimal bleeding.

Histologically dyskeratosis changes are seen top to bottom in the epithelium but basement membrane is intact. Treatment has been tried with cryosurgery, Diathermy, and application of cytotoxic drugs. Surgical excision of the entire lesion often is the modality of choice.

(*Note: Bowen's syndrome* is a Cerebrohepatorenal syndrome characterized by micrognathia, protruding tongue, high arched palate, elevated serum Fe levels, and depressed immunoglobulin levels—hepatomegaly and renal cortical cysts are the additional findings. This syndrome does not have any premalignant propensities.)

A benign variety of this disorder termed as Bowen's papullosis has been described which look quite fierce but is in reality benign. This label of Bowen's disease is used interchangeably with Erythroplakia, Erythroplasia of Querat, etc. in the oral cavity.

Dyskeratosis Congenita

Also termed as the Zinssner-Engman-Cole syndrome

This is a recessively inherited disorder seen almost exclusively in males. The oral changes in tongue and cheek show atrophic and leukoplakia like lesions, nails undergo bizarre aberrations, and skin shows reticulated hyper-pigmentation of the face, neck and thorax, leukemia and lymphomas are frequently associated with this disorder. Oral lesions start even before 10 years of age and malignancies supervene by early adulthood.

DLE—Discoid Lupus Erythematosus

The oral lesions of DLE are described as—circumscribed slightly elevated white patches surrounded by a red halo. A radiating pattern of delicate white lines surrounding the lesion is usually observed. It may also be seen as central atrophic area with white dots bordered with parallel line of striae. Majority of the oral lesions occur on cheek, gingivae and vermillion border of the lips. Burning sensation is a common feature. The premalignant potential of DLE is debatable and till it is resolved, it is wise for the clinician to treat it as pre-malignant. Topical and systemic corticosteroids are the drugs of choice.

Following drugs are known to precipitate a DLE like reaction in oral cavity

- Gold
- Isoniazid
- Phenytoin
- Tetracycline group
- Griseofulvin
- Streptomycin.

CONCLUSION

In developing country like India the role Dentists as primary health care providers with specially duty to detect oral precancer in early stage is becoming more and more important. Dental specialists of all specialties should make themselves familiar with the various lesions seeded with cancer potential which are becoming common in a wide spectrum of ages. Guptal PC et al[27] 1998 in their data drawn from the study in Palitana taluk of Bhavnagar district found that high fiber consumption and vitamin C both had protective effect on development of oral submucous fibrosis (OSMF) and leukoplakia . This indicates that apart from counseling the patients against the tobacco abuse it is a good idea to talk to them about their diet and its modification.

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Bailoor DN, Ani John

17 Oral Cancer: Squamous Cell Carcinoma

INTRODUCTION

Squamous cell carcinoma comprises of 90 to 95% of all oral malignancies. In this section we shall use the term Oral Cancer (OC) to be synonymous with squamous cell carcinoma.

In International Classification of diseases[1] by WHO 1977 Oral cancer is denoted with ICD 140 –Lip, 141-Tongue, 143-Gingiva and alveolar ridge; 144-Floor of mouth and 145-Other parts. In all centers in India we follow these recommendations meticulously. Incidence of OC in Asia ranges from 1.6 in Japan to 13.5 in India (Waterhouse et al).[2]

PC Gupta, A Nandakumar[34] (1999) in their editorial have stated that in India cancer of the oral cavity (C00.0 to C06.9, ICDO, 1990) is one of five leading sites of cancer in either sex. The age standardised incidence rates (ASR) vary from 6.5 per 100000 in Bangalore to 15.9 per 100000 in Trivandrum among males and from 7.2 in Bombay to 10.6 per 100000 in Madras among females.

In the Manipuri District of India annual incidence rate of 21.4/100, 000 has been determined by Wahi et al.[3]

In Ahmedabad India, Malaowalla[4] has established a crude incidence rate of 25 per 100,000 in his studies amongst the textile mill workers who were 35 years and above.

Relative frequency of OC in different countries in the Indian subcontinent is given in Table 17. 1.

Table 17.1: Relative frequency of oral cancer in various parts of Asia

Country	*Relative frequency*	*SC Worker*	*Year*
Bangladesh	18.4	Huq SF[5]	1965
Afghanistan	2.0	Sobin LH[6]	1969
Pakistan	18.2	Zaidi et al[7]	1974
India-Chandigarh	5.7		
India-Dibrugarh	14.7	Sanghavi et al[8]	1986
India-Trivandrum	20.2	Sanghavi et al[8]	1986

Table 17.2: Comparison of oral cancer in different parts of the world

Country	*Previous incidence*	*Recent incidence*
Puerto Rico	16.4	13.4
Finland	10.8	6.6
Cali, Colombia	8.4	5.1
Bombay, India	21.0	10.3

On observing the world data there seems to a perceptible decrease in the incidence of the OC in quite a few countries including India. See Table 17.2.

Conclusion of the table II may be explained by

- Increasing awareness of habit related risks and more propaganda of the tobacco abuse, consequent reduction in these habits.
- Better nutrition and living standards.
- Better early diagnosis facilities and increased governmental spending in the health sector and more health care facilities being available in the third world countries.

RISK FACTORS

The term risk factor appears to be more appropriate than etiology since despite voluminous statistics, few firm conclusions can be drawn as to direct cause of OC.

1. Tobacco Abuse: In Asian countries like India, *Bidi* smoking, Hookah, Clay pipe, Cigarette, Chewing tobacco or Smokeless tobacco and tobacco together with arecanut and lime are widely used from area to area. To quantify tobacco abuse Tobacco Index used by Author seems to have a strong correlation to dysplastic change in the oral mucosa. Details are discussed in white lesions chapter. Smoking and Chewing index above 100 appears to be strongly related with dysplastic conversion in South Kanara and North Kerala populations.

Silverman et al (1983)[9] discussed tobacco usage in patients with head and neck carcinomas, reporting that of 166 patients 73 percent used tobacco (90%) smoked cigarettes. These workers further reported that 30 percent patients who did not change their tobacco habits developed second primary cancers. Reduction or discontinuation of smoking appeared to lower the risk of developing a secondary primary cancer.

2. Areca Nut Chewing with Lime: It has a definite carcinogenic effect even in absence of use of tobacco. In India too, strong correlation has been shown between Betel nut chewing and Oral submucus fibrosis by Reddy R[10] and Balaji Rao.[11] Saman Warnakulasuriya.[35]

3. Alcohol abuse: Many studies all over the world have linked alcohol with Oral cancer, since most heavy alcohol users are also heavy smokers it seems difficult to independently assess the effect of ethanol. Rich and Radden[12] in their Australian study found 63% of their OC patients were alcohol users. McCoy[13] gave a possible mechanism by suggesting that alcohol alters the metabolism of oral and mucosal epithelium causing easy ingress for co-carcinogens; at a systemic level compromised liver function due to alcohol toxicity decreases the ability of hepatic cells to remove potential co-carcinogens from circulation. In our geographic area measuring Alcohol Index is done by classifying the alcohols into A1, A2 and A3 groups. A1 includes light alcohols. Beer and wine, A2 includes preparations like 'Gin', 'Rum', 'Whisky' etc. A3 includes very strong alcohols like "Arrack", "Patta" etc. which are frequently home distilled and unregulated amounts of additives and chemicals added. In our epidemiological studies there is a strong correlation between A1 and Leukoplakia of the floor of mouth and A3 and Oral cancer. Here too, we use an index to quantify alcohol abuse. Frequency of alcohol consumption per week multiplied by number of years, for example a laborer who consumes arrack four times a week for last ten years is denoted to have an index of A3-40 (4 times a week x 10 years = 40). In conclusion home brewed alcohol in heavy quantities appears to be a definite risk factor.

4. Diet and Deficiency States: The relationship between severe anemia and leukoplakia appears to be well documented. (Ranasinghe et al)[14] and so is the report by Lynch [15] which states categorically that of 250 patients of Ca of mouth and respiratory tract that 70% had plummer vinson syndrome (see appendix on syndromes).

Vitamin A deficiency causes epithelial atrophy and degeneration. So, it seems logical to conclude that diet is an important cofactor in development of malignancy in any part of the body. Diet high in fiber, fruits, salads, raw vegetables, low in fats-butter, red meat, high in carotene related products, appears to be the best protection against cancer.

Vitamin E appears to have some protective role in the carcinogenic process. The dental surgeon can give similar health advice to his high risk patients.

5. Actinic Radiation and Sunlight—causes the actinic keratoses lesions in the lips of the Indian farmers who work for long hours in the fields, but most of the times the melanin seems to protect the malignant conversion, the frequency of lip carcinoma is quite rare in the Indian Series see Table 17.3.

6. Herpes simplex virus and OC-Scully 1983[16] has reviewed the current knowledge concerning the association of herpes simplex virus and OC and concluded that cause and effect was not yet established.

7. Mouthwash: Long standing use of mouthwashes has been mentioned to be an risk factor for development of OC—Weaver and coworkers (1979).[17] AK Mascarenhas et al[40] has stated that Viadent® mouth rinse or toothpaste use is a risk indicator for oral leukoplakia.

8. Chronic Irritation: Older texts mention about the role of chronic irritation in the development of OC but now it has been confirmed that this alone cannot cause OC Spouge 1973.[18] This may be a tertiary implicating factor after tobacco and alcohol abuse.

9. Syphilis has long been associated with the Ca of the tongue, but with specific and effective antibiotic treatment being available the role of syphilis as an implicating factor in oral cancer is becoming redundant.

10. AIDS: This immunodeficiency syndrome is increasingly associated with various malignancies, including kaposi's sarcoma, squamous cell carcinoma and rare lymphomas. See the chapter on AIDS for more details.

11. Genetic Predisposition: Ankathil R et al in (1996) from Regional Cancer Center Trivandrum performed a detailed pedigree analysis and found that oral cancer tends to aggregate in family members even without tobacco and alcohol habits. They suggested that further inquiry into whether this could represent a site-specific autosomal dominant mode of inheritance must be questioned.

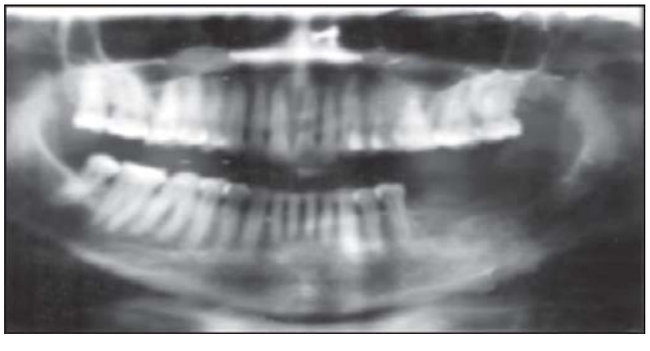
CLINICAL FEATURES

In India OC appears in one of the following forms in the oral mucosa:

- White patch like lesion with ulcerated area within or adjacent to it.
- As an ulcerated area with rolled borders and hard indurated edges, with velvety red irregular base. (Infiltrative variety) (Fig. 17.1B)
- As a proliferative growth with single or multiple ulcers around it, with induration (exophytic-verrucous variety) (Figs 17.3 and 17.5)
- Sometimes with white patch like lesion with interspersed reddish areas, which ulcerate (Fig. 17.2).

Age for this lesion is more common in 4th, 5th, 6th decade of life male to female ratios vary from 2:1 to 4:1, but with increasing number of women taking to smoking and drinking alcohol this ratio seems to be reducing. Regions that are most common include the buccal mucosa, the lower alveolus, floor of the mouth, lower lip and the tongue (Figs 17.1 to 17.5).

According to Khanna NN[19] if any of the above features especially the indurated edges, lymphnode involvement



FIGURES 17.1A to C: Shows extraoral, intraoral and radiographic pictures of a patient with squamous cell carcinoma of the alveolus (A&B). She had a extraoral swelling on the left lower border of the mandible since three months. She also had habit of chewing pan and keeping the quid in the sulcus, she hailed from north Karnataka where chewing habits are very prevalent. Radiograph (C) shows pathological fracture of the left angle of the mandible. (STAGE III T3 N1 M0) (Omal PM, Beena K, Bailoor DN 2003, Yenepoya Dental College Hospital, Mangalore)

FIGURES 17.2A and B: Shows extra and intra oral pictures of a patient with squamous cell carcinoma of the buccal mucosa. He had a draining extraoral sinus on the cheek since last one month, also had very high habit index. (Nillofer S, Prasanna K, Bailoor DN 2003, Yenepoya Dental College Hospital, Mangalore)

FIGURES 17.3A and B: Shows extra and intra oral pictures of a patient with squamous cell carcinoma of the floor of the mouth and alveolus. This was a 60-year-old female patient extra oral swelling since 4 months, she had severe pain and had weight loss. Intraorally a massive tumor involving the buccal mucosa, floor of the mouth and alveolus on the right side was present, she was referred to cancer institute for treatment (Nillofer S, Prasanna K, Bailoor DN 2003, Yenepoya Dental College Hospital, Mangalore)

and if tobacco and alcohol index is above 100 and 40 respectively, the clinician must harbor a high suspicion index and go for early biopsy.

One US study has shown a strong correlation of high rates of OC with the females working in apparel, textile and leather industries. Similar figures were also reported in the chemical and the paper industry (Blot and Fraumeni)[20].

Fifty-seven percent of the cases of OC appear in 5th and 6th decade of life, but now the trend seems to be that younger males appear to be involved; 11% of the Oral cancers are seen in patients below the age of 39 years (Sonis et al).[21]

Distribution of Oral Carcinoma according to site in American and Indian populations is shown in Table 17.3.

Table 17.3: Distribution of oral cancer in different sites of oral cavity in American and Indian population

American Population (Krolls and Hoffman)[22]		*Indian Population*		
Site	*%*	*Site*	*%*	*Reference*
Lower Lip	38%	Buccal Mucosa	45-80%	Hirayama[23]
Tongue	22%	Tongue	28-37%	Shantha[24]
Floor of Mouth	17%	Palate	32-48%	Reddy et al[25]
Gingiva	6%	Gingiva	2.6-12.6%	Wahi et al[3]
Others	17%	Floor of mouth and others	10%	Sanghvi et al[8]

Behavior of OC-squamous cell carcinoma; it spreads by local invasion and metastasize to the regional lymph mode via the lymphatic channels, severity of invasion is directly related to the degree of anaplasia. Distant metastases are rare.

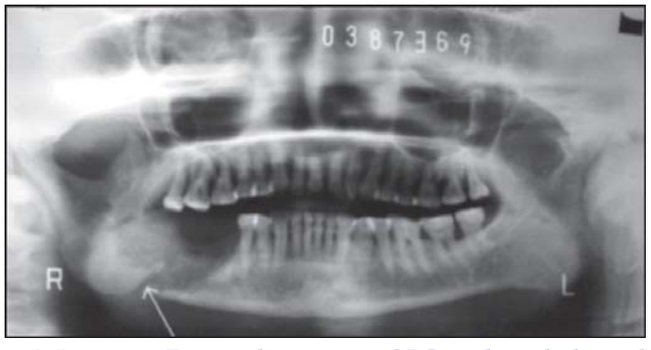


FIGURE 17.4: Figure showing an OPG with pathological fracture of body of mandible due to infiltration of intraoral squamous cell carcinoma (CourtesyKeerthilatha Pai, 2004 CODS Manipal)

STAGING OF ORAL CANCER**

See Table 17.4.

Rapidis et al[26] have modified the original classification given by AJCCS, i.e. the American Joint Committee for Cancer Staging and End Result reporting—where S Site; P pathological type of the tumor is added.

Evans et al[27] also agrees that this system of STNMP, which represents a considerable improvement in the prognosticating differentiation as, compared to the previous TNM. The latest edition of TNM classification is given in Table 17.4. In our departments now we use this system for recording of oral cancer patient data.

Table 17.4: TNM classification 6 th edition 2002

T	Size of the primary tumor
N	Nodal involvement
M	Distant Metastases
Tx	Primary tumor cannot be assessed
T_0	No evidence of primary tumor
T is	Carcinoma in situ
T1	Primary tumor = 2 cm
T2	Primary tumor > 2 cm and = 4 cm
T3	Primary tumor > 4 cm. Not fixed to under lying structures
T4	Primary tumor > 4 cm fixed to underlying structures
T4 a	Invasion through cortical bone into deep muscles/ Maxillary sinus/Skin of face
T4 b	Invasion in to masticatory space, pterygoid plates or skull base or encompasses internal carotid artery.
N	
N_0	No clinically positive nodes
N1	Single homolateral node = 3 cm
N2a	Single homolateral node = 3 cm but < 6 cm
N2b	Multiple homolateral nodes all < 6 cm
N3a	Homolateral node (s), at least one > 6 cm
N3b	Bilateral nodes
N3c	Contralateral nodes only
M	
M_0	N_0 known metastases
M1	Distant metastases present

Clinical staging	
Stage I	T1NoMo
Stage II	T2NoMo
Stage III	T3NoMo, Any T1, 2, or 3; N1, Mo
Stage IV A	T1,T2,T3 N2 M0 T4a, any N, Mo,
Stage IV B	Any T N3, Mo T4b any N M0
Stage IV C	Any T Any N M1

EVALUATION OF THE SUSPECTED CARCINOMA LESION

Once a lesion is detected based on the above mentioned clinical characteristics. All the details of history and

FIGURES 17.5A and B: Shows extra- and intraoral pictures of a patient with squamous cell carcinoma of the floor of the mouth. He had a very high habit index, with smoking, chewing of pan and alcohol intake (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College Hospital, Mangalore)

clinical exam should be written down in a format, photographs or color transparencies should be taken for subsequent comparison purposes. Lymphadenopathy details should be noted (Fig. 17.3).

Patient should be motivated to change his tobacco and betelnut chewing habit. Ill effects of abuse of raw alcohols must be explained in easy terms and visual aids like charts, slides should be used to motivate people with low level of education.

Leukoplakias in the oral region should be treated according to the treatment regimens mentioned in the chapter on leukoplakias.

Probable source of irritation should be reduced or eliminated when possible. An observation appointment for re-evaluation of the lesion should be scheduled 10 to 14 days later. If resolution of the lesion occurs in this time, further observation is done, otherwise this lesion must be considered high risk and immediate biopsy should be attempted.

In the villages where definitive treatment for the oral cancer is not available, it is advised that the dental surgeon keep formalin or absolute alcohol to preserve the biopsy specimens in good condition as he sends to the nearest teaching/research center. Moreover he must not refer the

Oral cancer case to a distant center purely for clinical diagnosis. Most of the times a histopathological report will be available between 10 and 15 days even considering the postal delay!

Methods that are used to help in diagnosis are as follows:

- Exfoliative cytology-good mass screening technique but low specificity.
- Toludine Blue staining
- Toludine Blue + Lugols Iodine method
- Incisional/Excisional biopsy method *Routinely recommended
- FNAB or Fine needle aspiration biopsy method (inaccessible areas).
- Brush Biopsy or OraScan® this service is used in the US and not yet available in India. A special brush is provided which is used to scrape the lesion and then transported to a computerized center via special sealed tube. Computer looks at digital images of the cells and then using a expert system program gives a fairly accurate assessment of the condition of the cells.
- Vizilite®: Vizilite is a simple medical device to improve the identification, evaluation and monitoring of oral mucosal abnormalities in populations at increased risk for oral cancer. Oral dysplastic soft tissues exhibit characteristic features following an acetic acid wash and visual inspection under chemiluminescent illumination. Termed as 'aceto-white' by Huber MA et al.[41] This principle has been used by Vizilite® manufacturers and it is being marketed all over the world by Zila, Inc. 5227 North 7th Street Phoenix, AZ 85014-2800. ViziLite is the only medical device available for early detection of oral cancer. ViziLite empowers the practice to provide its patients the most comprehensive oral cancer screening possible.

Toludine Blue Staining

Topical application of the staining medium to the oral mucosa is followed by rinse of 1.0% acetic acid (Table 17.5). The dye, retained predominantly in the abnormal nuclei of the tumor cells, produces areas of uptake seen as directly blue stained tissue. Rinsing is performed to remove dye retained by debris or within irregularities of the mucosal surface. Biopsy must be invariably performed in those areas where uptake is positive.

Mashberg[28] studies indicates that the toluidine blue stains has some efficacy for the early detection of certain oral lesions including squamous cell carcinoma, when used in conjunction with other diagnostic techniques.

Exfoliative Cytology

Smear techniques have a good screening utility and normally do not have much application in the Dental Clinics because they are non-conclusive and negative smear report does not have much value. If positive still, biopsy needs to be done.

Epstein et al[29] have mentioned the use of Lugol's iodine and toludine blue together and stated that this combination of the stains was very useful in delineating the normal and the dysplastic tissue. Lugols iodine stains the normal mucosa brown and the toludine blue is retained in proportion of the abnormality. The color contrast provided makes it easier for the practicing dental surgeon to identify the biopsy site.

Formulation of the tissue stain can be done as follows:

Table 17.5: Depicting the constitution of Toludine Blue and Lugol's Iodine solution

Toludine Blue solution	Lugol's iodine solution
Toludine blue 1 gm	Iodine 2 gm
Acetic Acid 10 cc	Potassium iodine 4 g
Absolute alcohol 4.2 cc	Distilled water 100cc
Distilled water 86 cc	
PH adjusted to 4.5	

The method of application

Isolate and dry the area, which has the lesion, and apply 1% acetic acid with a camel hair brush (wait 20 secs)

- Rinse with water
- Apply Toludine blue 1% with fresh brush (wait 10-20 secs) Decolorise with 2% acetic acid
- Apply Lugols iodine
- Photograph the area. Plan biopsy in such a way that half the incisional biopsy should contain the normal, i.e. Lugol's iodine stained tissue.

Biopsy Procedure

Once the lesion is identified using any of the staining methods, following method could be used to do an incisional biopsy (Fig. 17.6).

- Suitable block LA should be attempted using lidocaine with epinephrine—Lignox® 2-4 cc according to the size and location of the lesion.
- 000 silk suture is introduced into the tissue of interest.
- This suture is used to elevate the lesion and an elliptical incision should be made around it such that at least 33% of the normal area could be included.
- Once the specimen is removed it should be gently washed in flowing water and slowly teased into 10% Formalin.
- Formalin 10% should be at least 10 times the volume of the tissue mass. Absolute Alcohol may be used if Formalin is unavailable.
- A concise history, clinical features and your clinical diagnosis and additional data, hematological, radiographic or any other if available should be provided; to the Oral Pathologist which will assist him to make a accurate diagnosis. Biopsy report received should be Xeroxed and copy maintained in the files of the clinic, before the patient is referred to another specialist such as Oncologist.

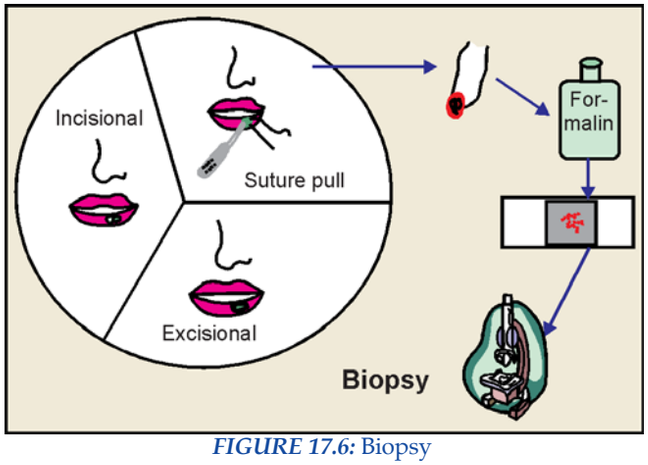


FIGURE 17.6: Biopsy

TREATMENT

Oral cancers are generally treated with

- Surgery alone
- Radiation therapy alone
- A combination of surgery and radiation
- Chemotherapy
- Gene Therapy/Immunotherapy has been tried at selected centers and is normally used in conjunction with either one of the above.

Chemotherapy is used either in adjuvant setting or for palliation

- Cryosurgery
- Hyperthermia

Small lesions such as Stage I and Stage II (Refer STNMP classification table No. IV) Carcinoma is typically treated with surgery alone, or radiation alone, Stage III and Stage IV are treated with surgery followed by radiation. When lymph nodes are involved radical neck dissection of the affected side is attempted concomitantly. Radiation dosage either primary or adjunctive ranges from 4000 to 7500 rads which is delivered over 6 to 7 weeks.

Surgical Treatment

Small lesions are excised leaving a 1 cm margin around the lesion and large may require a Commando type operation.

Radiation Therapy

Ionizing radiation is an effective modality for the treatment cancer of the oral cavity.

Brachytherapy and Teletherapy are the two types.

Brachytherapy involves use of Radon seeds, Radium needles or Iridium wire, Tantalum plates/wires etc.

Teletherapy could be given using Cobalt 60, Cesium units or Linear Accelerators. The latter are now being extensively used since they achieve shorter treatment time, deeper and more homogenous tissue penetration, creating of sharper field margins and sparing the overlying skin and underlying normal tissue from the radiation damage. It can be used alone for cure of small lesions (T1 or T2) or as a part of combination with surgery in larger lesions (T3 T4) administered preoperatively or post-operatively—Theodore et al.[30] It can also be used for pallitation. (Refer chapter no.40 on radiotherapy for detailed description).

Chemotherapy

Several drugs are effective when used singly but the response rates are low (20-40%) and the response duration

short. Methotrexate as a single drug and in various dose schedules has been extensively used.

- Cisplastin has been found to be an effective drug in oral cancer with the response rate of about 25%.
- Bleomycin is particularly effective against squamous cell carcinoma of the oral cavity. 5-fluorouracil has been successfully used in a number of combination chemotherapy regimens (Table 17.6).

Table 17.6: Response rate of combination chemotherapy in OC

Investigator	*Drugs*	*Response rate (%)*
Price[31]	Vincristine, Bleomycin methotrexate, 5-Fluorouracil	67%
Khanna[32]	Bleomycin, Methotrexate 5-Fluorouracil	79%
Weaver[33]	Cisplatin + 5-Fluorouracil	93%

Lasers in Cancer Therapy

Advantages of CO_2 laser include precise excision with microscopic control, minimal blood loss, the sealing of lymphatics, possibly decreasing the tumor cell spread and decreased postoperative edema. See Chapter No 34 on Lasers for details.

Photodynamic therapy kills the cancerous tissue and the precancerous tissue, but not the normal tissue. Other laser treatments destroy cancer cells by heating them or cutting them out along with healthy tissue. Photodynamic therapy uses a laser to produce a chemical reaction that kills the cancer cells without harming healthy tissue.

Kubler AC et al[39] have stated that Foscan-PDT yields complete response rates comparable to those published for surgery or radiotherapy without causing major toxicity. It allows preservation of form and function and does not compromise future treatment options for recurrent, residual or second primary disease. They used Foscan-mediated photodynamic therapy (Foscan-PDT) in patients with primary cancer of the lip, 0.15 mg/kg intravenously, followed by a single non-thermal illumination of the tumor 4 days later. Response was determined after 12 weeks.

Cryosurgery and Hyperthermia

Cryosurgery and hyperthermia are infrequently used as primary treatment measures in management of OC. Cryosurgery involve use of liquid nitrogen in the sudden death of the tumor cells. It is most frequently used for control of early lesions in debilitated or in palliation of non resectable lesions. Hyperthermia has been used in conjunction with the Radiation or Chemotherapy regimens and they are used in very few centers around the world.

Gene Therapy

Gene therapy for oral cancer is currently under investigation in clinical trials. The goal of cancer gene therapy is to introduce new genetic material into target cells without toxicity to non-target tissues[36] Adenovirus (Ad) vectors are commonly used in gene therapy trials because of their efficiency in gene transfer.

Rudin C et al[38] used ONYX-015 a genetically engineered, or altered, adenovirus (Advexin®). This virus has been altered in such a way that it will infect cancer cells, ultimately killing them, but will not infect normal healthy cells. ONYX-015 was developed to specifically target and infect cells with a destroyed or mutated p53 gene. In this clinical trial they evaluated a mouthwash that contains Advexin® in patients with precancerous areas of the mouth. A mouthwash has advantages over systemic (full body) therapy in that it is confined to the local area of concern, which potentially improves the rate of tolerability of treatment. They found favourable results with regession of the lesion in more than 50% of cases.

Nishikawa M[37] in an experimental study have showed that suicide gene therapy combining herpes simplex virus thymidine kinase gene (HSVtk) and ganciclovir (GCV) is one strategy for the treatment of head and neck squamous cell carcinoma (HNSCC) that led to apoptosis of the oral squamous cell carcinoma cell line.

Immunotherapy/Biotherapy

Immunotherapy/biotherapy is designed to repair, stimulate, or enhance body's own immune responses. Treatments such as interferon and colony stimulating factors are used either alone, or in conjunction with other modalities such as surgery, radiation and chemotherapy for better prognosis.

Biotherapy may be used to:

- Stop, control, or suppress processes that permit cancer growth;

- Make cancer cells more recognizable, and therefore, more susceptible, to destruction by our immune system;
- Boost the killing power of immune system cells, such as T-cells, NK-cells, and macrophages;
- Alter cancer cells' growth patterns to promote behavior like that of healthy cells;
- Block or reverse the process that changes a normal cell or a precancerous cell into a cancerous cell;
- Enhance body's ability to repair or replace normal cells damaged or destroyed by other forms of cancer treatment, such as chemotherapy or radiation; and
- Prevent oral cancer cells from spreading to other parts of body

Role of Dental Surgeon in the Management of OC

The management of OC in India could be depicted in form of a diagnostic chain. This chain has three links.

- The well informed patient, who visits the dental surgeon early at the first sign of a white lesion
- An alert dental surgeon who diagnoses the OC in its early stages
- A competent Oncologist—who with help of tumor board decides the treatment to be given to the patient.
- The use of Toludine Blue and Lugols Iodine staining and subsequent biopsy procedure.
- Extracting all the periodontally affected teeth in the line of the radiation and all the teeth, which may cause secondary irritation.
- Taking patients photographs, radiographs, impressions, both dental and facial if major surgeries are contemplated so that the prosthodontist can reconstruct the facial structures successfully.
- Role of dental surgeon in managing the radiation mucositis, Xerostomia, mucosal ulceration, and osteomylitis is as important as the primary treatment by the oncologist.
- Post treatment oral monitoring includes controlling infection using 0.12%. Chlorhexidine and various antifungals, topical fluorides to prevent cervical caries, dietary management.
- Dental surgeon must liaison with the clinical psychologist to manage post treatment depression which is present in large number of patients and specially in those who have significant facial disfigurement.

CONCLUSION

Apart from being the guardian of dental health of the patient, it is the dentist's duty to keep a sharp eye for the premalignant lesions and conditions. He also plays a stellar role in the education of the patient in the ill effects of tobacco, raw alcohol and chewing of betel leaf, betel nut in various combinations including the smokeless and the snuff variety. Dietary counseling by dental surgeon should include—

- Reduction in the spiciness, Chilli in the routine diet.
- Increased amount of 'fiber' in form of salads, raw fruits, etc.
- Increase the intake of the protective micronutrients like Vitamin A, Vitamin E, Betacarotene, and Vitamin C all of which are supposed to protect the oral mucosa by taking fresh fruit—papaya, oranges, lime juice, or in form of vitamin tablets which are economically feasible for the poor patients of Indian villages as well as which will give consistent level of ingestion.
- A conscious reduction in fat from the diet resulting in the reduced consumption of butter, egg yolk and red meat.

The effective management of an oral cancer case depends on the three factors "Three-link theory" (see Fig. 17.7).

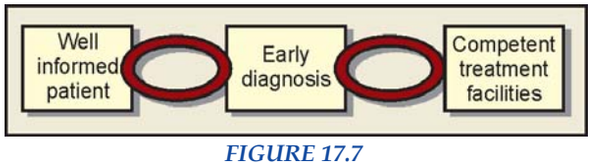


FIGURE 17.7

Dentist Educates = Dentist Recognizes Precancer= Dentist is part of Oncology team.

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Bailoor DN, Nagesh KS

18 Salivary Gland Disorders

INTRODUCTION

The human beings are blessed with three pairs of major salivary glands and many millions of minor salivary glands. They secrete saliva, which helps in lubrication, in taste perception and in overall protection of teeth and Oral mucosa through the Immunoglobulins and other antibacterial factors. These glands are prone to a special set of diseases, which will be touched upon in this section.

When a Dental patient presents with one of the following chief complaints-

- Swelling unilaterally or bilaterally in one or more of the salivary glands.
- Pain and swelling increasing during chewing food, or even viewing food.
- Lobulated swelling in one of the glands.
- Dryness of mouth and eyes with joint pains.
- Xerostomia, partial or complete. We try to evaluate for Salivary gland pathology.

Navazesh[8] mentions four clinical measures to diagnose the hypofunction in the salivary gland. Dryness of the lips, dryness of the buccal mucosa, absence of saliva produced by gland palpation and the DMFT scores. He mentions the normal salivary flow rates to be 0.12 to 0.16 ml/min.

INVESTIGATIONS

The evaluation could be done in:

1. Plain films
2. Sialography
3. Special imaging
4. Sialochemistry

Plain films indicated in this region are occlusal, PA mandible with cheek blown out, and lateral jaw projection with patients finger depressing the tongue and consequently the floor of the mouth, all provide for the basic scout films. Gross calculus can be viewed in such projections but fine dystrophic calcifications require CT scan, since the sensitivity is increased 10 times, in that imaging technique.

Sialography

Definition

It is a specialized radiographic view taken by introduction of the radiopaque dye into the ductal system of the major salivary glands, mainly parotid and submandibular. The sublingual and the minor glands cannot be studied obviously because of their small and numerous openings.

Indications

1. Detection of Sialoliths both radiopaque and radiolucent.

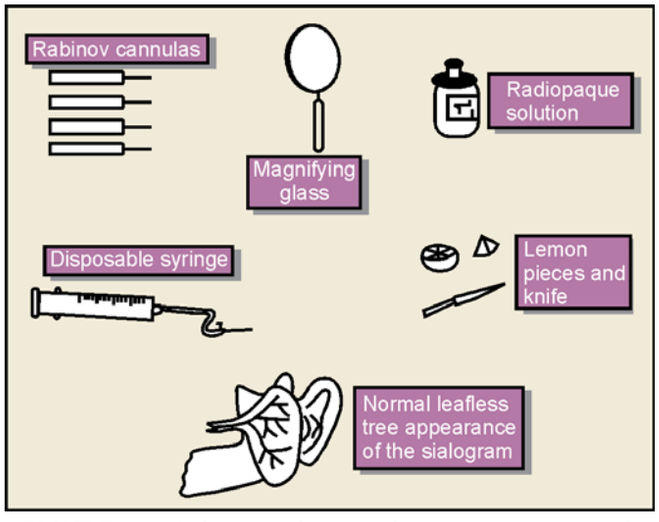


FIGURE 18.1: A diagram showing the equipment required for sialography

2. Evaluation of the extent of irreversible ductal damage caused by infection.
3. Differentiation between the Sjögren's syndrome, Sialosis and chronic sialadenitis.
4. Evaluation of diverticula, strictures and fistula.
5. It may be used as a dilating procedure for mild ductal stenosis.

Contraindications

1. Acute infection of the salivary gland.
2. Allergic reaction to any of the components of the radiopaque material to be used.
3. Thyroid disorders in patients due to the Iodine content of the contrast media.

Armamentarium

1. Sialographic Cannulas-Rabinov Cannulas-with tips ranging from 0.012 to 0.033 inches.
   Most of these cannulas come with polyethylene flexible tube.
2. Lacrimal Dilators ranging from 0000 to caliber 0.
3. A 5 ml syringe (disposable)
4. Gauze sponge pads
5. Conray 420, Urograffin
6. Secretogogue such as fresh Lemon, Lemon extract or lemon concentrate.
7. Good dental lighting.
8. Magnifying glasses

Method

Once the duct is cannulated the injection is made with hand pressure. The patient may complain of mild pain during at injection, however, a slow constant injection technique usually can accomplish complete ductal filling without much patient discomfort. The patient's sensation of glandular fullness is suggested by a sharp pain when the operator usually stops and proceeds with radiography.

Interpretation

The sialographic appearance of the normal salivary gland is that of a leafless tree. This radiograph shows the main duct gradually going in secondary branches and then into tertiary branches.

Chronic Recurrent Sialadenitis: Shows focal narrowing of the main duct and central ductal dilation (Sialectasia), these dilated ducts often taper down dramatically to normal peripheral ducts.

Sjögren's syndrome usually results in the punctate filling defects initially and then mulberry tree appearance on the sialogram (Fig. 18.1).

Sialographic Findings

Sialography is an invaluable asset in the diagnosis of neoplastic diseases of salivary gland origin. When the tumor is clinically manifest, the sialogram may reveal positive changes when studied by a trained observer. Unfortunately, the changes produced by the small neoplasm or by tumors in an early stage of development are too subtle to be clearly detected by this technique, particularly when they are peripherally located. However, deliberate over filling to obtain acinar shadows may be of value in such cases. Encountering a peripherally located radiolucent indentation upon the opaque shadow of the gland may serve to identify the lesion. Since the benign tumor develops at the expense of normal glandular structure, the sialogram will often reflect its presence by revealing a filling defect, the latter being due to distortion and displacement of the normal duct system by the pressure of the expanding mass.

A centrally located defect, devoid of ducts and surrounded by a whorl-like formation of ducts, is referred

to as the "ball-in-hand." The tumor with no ductal structures in its midst represents the "ball" whereas the normal secondary and tertiary ducts that have been pushed to the periphery are supposedly the fingers and palm of the "hand". This pattern may be visualized on lateral and/or anteroposterior films. Despite the distention of the individual ducts, the duct system is intact and, unless the tumor is exerting severe pressure, which in turn may lead to duct obstruction, emptying of the sialographic solution occurs within a normal period.

Noting the presence of localized puddling or widespread diffusion of the contrast medium thought the gland parenchyma should suggest the diagnosis of a malignant neoplastic disease. The invasive character of the malignant tumor leads to partial destruction of ducts, and as the sialographic solution reaches these regions, it escapes into the surrounding interstitial connective tissue, either accumulating in localized puddles or diffusing widely.

In such cases, emptying film studies indicate retention of contrast media. Not all malignant tumors are portrayed in this manner. Occasionally, a malignant neoplasm gives the "ball-in-the hand" pattern, since it, too, may manifest a tendency to encapsulation in spite of is infiltrative character.

When dealing with a parotid gland tumor, an anteroposterior film may shed further light regarding its nature and more precise location. The parotid gland with its contained duct system may be displaced laterally away from the ramus of the mandible. Thus, the exact location of the lesion is noted and its size may even be precisely measured.

Displacement of Stensen's or Wharton's duct by the presence of a tumor may also be detected sialographically. With forward displacement of the gland, buckling of the major duct is observed, with the posterior portion crowding upon its anterior segment. Posterior glandular displacement results in the opposite effect—a distention and elongation of the major duct. In addition, inferior or superior gland displacement inevitably causes a disturbance in the course of Stensen's and Wharton's ducts and this, too, may be visualized sialograhically.

Special Techniques

Garc´ia CJ et al (1998)[28] mentioned that Ultrasonography (US) should be the initial imaging study used for the examination of salivary gland lesions in children, given the fact that most of such lesions are benign and are shown up clearly by sonography. In most cases, this technique permits the differentiation of intra-glandular and extra-glandular lesions. Vascular lesions can be demonstrated more clearly through the use of color Doppler imaging.

Nahlieli O and Baruchin AM (1999)[30] studied about Sialoendoscopy in 154 salivary glands. They have given 4 indications for endoscopy.

1. Calculus removal that could not be performed by conventional methods,
2. Screening of the salivary ductal system for residual calculi after sialolithotomy
3. Positive evidence of ductal dilatation of stenosis on the sialogram or ultrasound examination,
4. Recurrent episodes of major salivary gland swellings without known cause. Sialoendoscopy is a minimally invasive technique which works fine in many obstructive disorders of salivary glands.

Ohbayashi N et al (1998)[32] found that magnetic resonance (MR) sialography is highly accurate in the evaluation of salivary gland disease in Sjögren syndrome.

CT and MRI are the examinations of choice for evaluating the mass lesion. They can identify the presence of a mass, its location and its position relative to the facial nerve. The salivary gland has normally fatty interstitial structure the CT attenuation of the gland is 25 to 15 Hounsfield Units. HU is lower than that of muscle, but more than that of fat.[2]

Sumi M et al (1999)[17]their study suggests that MR imaging features may reflect chronic and acute obstruction, and a combination of CT and MR imaging may complement each other in examining glands with sialolithiasis.

Sialochemistry

It is the laboratory analysis of saliva which assists in diagnosis of salivary gland pathology and in some cases other pathology. The science of sialochemistry is not yet fully developed but the current trends indicate that in the

coming decade saliva analysis will become as important as serum analysis.

Wang S et al (1996)[10] analyzed saliva of 28 patients with Sjögren's syndrome (25 of obstructive parotitis, 32 with sialadenosis and 32 normals). They found that in SS group, saliva flow rate was decreased concentration of SIgA, IgG, electrolytes was significantly elevated. In the COP group saliva sodium, potassium, chlorine and calcium were decreased. In the sialadenosis group phosphorous was found to be elevated. They have concluded that the total value of immunoglobins and electrolytes has greater value than that of concentration in diagnosis of various disorders.

Gonz´alez M et al (1997)[11] stated that salivary monitoring could be used for detection of environmental pollutants, drugs, etc. Heavy metal pollution continues to be a public health problem, and sialochemistry seems to be an easy way of assessment for large populations. In their study of saliva no association was found between the variables; age, sex, geographic area and canned food consumption with Pb and Cr. However, an inverse association was found between Cd and age.

Banderas-Tarabay JA et al (1997)[12] studied the salivary flow rates and total protein concentrations by gravimetric and spectrophotometric analysis. They found that they would be able to establish a sialochemistry database for population of mexico through their studies.

SIALODOCHITIS FIBRINOSA (KUSSMAUL'S DISEASE)

It is characterized by painless recurrent attack of submandibular or parotid gland swelling secondary to a fibrinous ductal plug.

Work et al (1973)[6] mentioned that this condition is seen in the dehydrated and debilitated patients. The treatment is glandular massage and use of Secretogogues to release the plug.

SIALOLITHIASIS

Whenever the salivary duct or the gland proper is blocked due to the formation of a calcified structure it is termed as salivary stone or sialolithiasis. It may affect major or minor salivary gland.

The stone appears to have a laminated structure with concentric layers of calcifications around a nidus or a central portion. Chemical structure is mainly Calcium phosphate with traces of magnesium, potassium chloride and ammonium. The occurrence of the Sialolith is about 80 percent in the submandibular gland duct and 18 percent in the parotid and 2 percent in the sublingual and minor salivary glands. 1/5th of all cases have multiple calculi. Chief complaint of the patient is mainly pain in the major salivary gland area and complaint of swelling on seeing food or just on sitting for meals. This pain is due to stasis of the saliva and later this may lead to infection and painful swelling and redness over the affected area.

Sialoliths are more in the Wharton's duct because it is more convoluted, the saliva has to travel against gravity and the viscosity of the saliva in this gland appears to be more. In the minor salivary glands if blockage occurs it presents as a freely movable draining swelling of the area (Fig. 18.2).

FIGURE 18.2: Figure showing occlusal radiograph of Sialolith of submandibular duct (Courtesy: Borle Rajiv et al , Sharad Pawar Dental College, Wardha 2003)

Diagnosis

1. Bidigital palpation
2. Occlusal or Posteroanterior radiograph of the jaws.
3. Sialographic demonstration of the sialolith.
4. Sialoendoscopic evaluation of ductal system.

Diagnosis of this Sialolith requires its actual palpation by bidigital method and visualization by the occlusal film,

that is well positioned. 1 in 5 sialoliths may not have enough calcification to be seen in a radiograph. In such cases the only way to demonstrate it would be the Sialographic examination or endoscopic visualization. Anteroposterior view of the face also demonstrates the parotid sialoliths in some cases.

Treatment

Management of sialolithiasis includes treatment of acute infections with antibiotics like Capsule Cloxacillin, or combination like Ampiclox, for period of 7 to 11 days appear to work well. Pain is controlled best by giving Ibuprofen + Paracetomol combinations (Imol, Ibugesic, etc). Concomitant use of lieces of lemon or other food substances to stimulate the salivary flow often displaces the salivary plug or small sialolith.

Stones in the distal portion of the duct may be manually removed by the dilators itself; if this is not possible, surgery is performed. In cases of repeated sialoliths surgical excision of the gland becomes mandatory.

Yoshizaki T et al (1996)[20] have reported treatment of Sialolith using extracorporeal shock wave lithotripsy (ESWL).

Since 1980, extracorporeal shock wave lithotripsy (ESWL) has been in clinical use for the treatment of renal and gallbladder stones. They used this technique as a treatment for sialolithiasis on 15 patients. They concluded that the ESWL successfully treated most sialoliths with minimum side effects.

Arzoz E et al (1996)[19] describes a technique of salivary gland endoscopy which allows intracorporeal lithotripsy under endoscopic control. Two types of energies were used, first Laser energy and second Pneumoballistic. The use of endoscopes with a working channel allows irrigation to improve visibility during exploration. In this series, pneumoballistic energy (Lithoclast) has been shown to produce calculus fragmentation with more efficiency than lasertripsy (Dornier Impact). When dilation and placement of a cannula (Abocath 16 G) was done 2 days preoperatively, endoscopy was performed more easily.

Yoshino N et al[15] 1996 have clarified the usefulness of Dormia basket, a stone retrieval catheter with fluoroscopy. They call it interventional radiology for sialolithiasis. Of the 16 patients treated, 10 were treated successfully and four could not get the calcified structure out due to its attachment to the duct and another two were unreachable. They conclude from this series that interventional radiology is less traumatic than routine surgery and should be first treatment of choice.

Nahlieli O et al (1997)[16] describes successful use of sialoendoscope for treatment of sialolithiasis in 46 major salivary glands. Of these only four endoscopic examinations failed due to technical reasons and all the rest were able to detect and extract the salivary stones in the ducts. They opine that this is a minimally invasive technique for calculus removal and a good diagnostic procedure.

Guiral H et al (1998)[18] have described an uncommon case of infection called actinomycosis caused by *Actnomyces israelii* noted in submandibular sialolithiasis. They emphasize that bacteriological analysis of all stones is a good idea.

Zenk J et al (1999)[13] analyzed data from 635 patient histories and follow-up examinations of sialolithiasis (SLT). SLT predominated among age of 30 and 70 years with no sex predilection. Submandibular ducts accounted for 78.9% and parotid glands for 21.1%. A simultaneous stone disease of the urinary tract or the bile duct system occurred by chance (4.3%). Sonography is the first choice of imaging.

Riesco JM et al (1999)[14] studied the Sialolith by microscopy and scanning electron microscopy and found that this stone had no concentric laminar structure. X-ray microanalysis revealed a high content of Sulphur followed by Calcium, Silicon and Sodium. The results suggest that the sialolith was young and later would undergo laminar maturation.

AUTOIMMUNE SIALOSIS

In 1892, Mikulicz disease described peculiar chronic symmetrical enlargement of the salivary and lacrimal glands.

Subsequently Gougerot, a French skin specialist described a condition of Lacrimal and Salivary gland swelling with infiltration of lymphocytes associated with Xerostomia Keratoconjunctivitis sicca. Henrik Sjögren a Swedish ophthalmologist reported the disease in detail and mentioned systemic manifestations.

Morgan et al (1953)[7] in his work found that these diseases mentioned historically above were in fact the same. This has led to a tendency to call this syndrome as Mikulicz-Gougerot-Sjögren's Disease. (MGJ Disease) In recent years, however, we use the term Autoimmune sialadenitis to characterize this disorder.

Asmussen K et al (1996)[22] studied the clinical features of 80 patients with primary Sjögren's syndrome (PSS). They found 3 tests related well with the extent of clinical disease. The level of immunoinflammatory activity (assessed by plasma IgG, serum ANA and focus scoring of minor labial salivary gland biopsies).

Fox RI (1996)[24] mentions that Primary Sjögren's syndrome (SS) is a systemic autoimmune disease that is characterized by keratoconjunctivitis sicca and xerostomia due to lymphocytic infiltrates of the lacrimal and salivary glands.

It is important to distinguish SS (an idiopathic autoimmune process) from other processes, including hepatitis C infection, autonomic neuropathy, and drug side effects.

Gobetti JP et al (1997)[23] mention that the secondary effects of xerostomia are very important for the dentists. They are candidial infection, caries and inflammation of oral mucosa. All caused by secondary effects of xerostomia.

Fox RI et al (1998)[25] differences in diagnostic criteria for Sjögren's syndrome (SS) have led to confusion in the research literature and in clinical practice. A particular challenge is the clinical diagnosis of the patients with sicca symptoms, fibromyalgia, chronic fatigue, vague cognitive defects, and a low titer antinuclear antibody. Until recently, many of these patients would have been classified as primary SS using the European criteria. A suggested revision of the European criteria will require inclusion of anti SS—A antibody or characteristic minor salivary gland biopsy, leading to greater agreement between European and San Diego criteria.

Davidson BK et al (1999)[21] have followed up 100 patients of MGJ over 10 year period. Although primary Sjögren's syndrome is often a benign condition, characterized by lymphocytic infiltration of salivary and lacrimal glands, some patients develop systemic features. Both HLA B8 and DR3 were present in 79% of Ro/La-positive. They have found that anti-Ro antibodies identified patients with more systemic disease, with increased incidence of parotid swelling, lymphadenopathy and lymphoma. Patients who are initially autoantibody negative, (including Ro and La negative) do not evolve into 'systemic' Sjögren's syndrome or other connective tissue disease.

Clinical Findings

The average age of 3 to 4 years the children appear to show the signs of recurrent parotitis, with male predominance. Sudden swelling of the parotid gland is seen mostly bilaterally. Recurrent changes in sizes, decreased salivation, partial xerostomia are seen. The frequency of recurrences increases during childhood and may or may not diminish with puberty. Varying degrees of parotid enlargement may persist between the episodes. There is a concomitant decrease in salivary flow. In addition, the quality of the saliva may be altered.

This clinical picture may be confused for infective recurrent parotitis in children. However, the child is not ill, although an increase in the number of the white blood cells may be found. Fever may occur but when present it appears only at the onset of the parotid swelling. Mild pain is experienced during an attack.

Protein studies are of significance. There is a moderate rise in total serum protein, the serum albumin level is normal and there is an elevation of the serum globulin level, usually resulting from an increase in the gamma fraction.

Recurrent parotitis in adults is not significantly different in its clinical picture from its counterpart in children except that the reported incidence is greater in women than in men.

An occasional involvement of the submandibular salivary glands has been found.

There is a great tendency for secondary infection; frank pus emerging from the ductal orifice and acute subjective symptoms affirm the infection. The relatively common finding of infection in adults suggests that progressive gland degeneration, over a period longer than that experienced by children with recurrent parotitis, predisposes the parotid gland to more severe infectious insult.

It is possible that the adult variety of recurrent parotitis in some patients represents a progression of the disease from its childhood form. Of particular interest is that in both varieties, Mikulicz disease or Sjögren's syndrome may have been present before the more unique traits of the disorder were manifested.

In many cases several adult patients who initially have only parotid swelling, but who develop the systemic signs of Sjögren's syndrome.

Mikulicz's disease, since the microscopic picture is identical and clinical symptoms approximate each other, no attempt will be made in this text to separate Mikulicz's disease from Sjögren's syndrome. It is sufficient to say that their differences rest in the fact that Mikulicz's disease represents a more local disorder occurring in both males and females, whereas Sjögren's syndrome is a systemic disease, confined predominantly to females.

The symptoms in patients with MGJ disease fall into two broad categories—*inflammatory-obstructive* and "neoplastic simulating".The inflammatory-obstructive group is characterized by glandular swellings which fluctuate in size with intermittent remissions. The neoplastic-simulating type usually is accompanied with a history of a long, gradual increase in gland size with no fluctuations.

In Sjögren's syndrome a Triad of symptoms-Rheumatoid Arthritis, Keratoconjunctivitis sicca and xerostomia with or without salivary gland swelling is noted. Occasionally the Rheumatoid arthritis is replaced by Lupus Erythematosus, Scleroderma, Polymyositis, Periarteritis nodosa.

Rough determination of the Lacrimal secretion may be obtained by the Schirmer Test.

- ***Slit Lamp examination*** for the presence of punctate corneal stains after the use of Fluoresecein or Rose Bengal gives more accurate results. Such stained areas denote corneal damage from inadequate lacrimation.
- ***Salivary flow rate*** can also be used for the scientific determination of the partial xerostomia. Burning of the mouth frquently results due to this dryness.

Denture wearing is uncomfortable. In dentulous patients cervical caries resembling the radiation caries results often.

Ascending infection of the salivary glands often results due to the salivary lack. Saliva then becomes flocculent and pus filled. Fever, Leucocytosis etc. are seen.

Sciubba JJ (1994)[9] have outlined how salivary dysfunction can cause wide array of changes in oral mucosa and dental structures. Treatment of Sjögren's syndrome should aim at restoring the taste, masticatory and protective functions by use of artificial saliva, fluoride application and oral hexidine application.

- ***Rheumatic factor*** is ascertained by Latex fixation and Bentonite Flocculation. Blood picture sometimes may show Eosinophilia (above 3%) Leucopenia (below 4000 cells per cc) and Thrombocytopenia (below 150,000 cells per cc) Hepatosplenomegaly and Acholorohydria have been also strongly associated.

Sialographic findings:

Sialographic studies in patients with autoimmune sialosis are significant in that sialectasis is a common denominator. Nevertheless, many patients show either a normal duct arborization or only slight atrophy and thinning of the individual ducts. It is possible that in the early stages of sialosis few changes are apparent sialographically. As the disease progresses, however, the appearance of the characteristic sialectic pattern is expected. Both parotids are involved in varying degrees. At times, the submandibular salivary gland may demonstrate similar changes. When present, sialectasis is observed in one of four increasingly severe stages—punctuate, globular, cavitary and destructive. Progression through the various stages has been observed in adults, but only the punctuate and globular patterns are found in children.

Punctuate sialectasis refers to accumulations of contrast solution of less than 1 mm in diameter. It is now known that the pooling of opaque medium is a result of intralobular duct dilation behind the impediment caused by hyperplasia of ductal epithelium, with narrowing of the lumen, rather than by an extravasation of fluid into the periductal connective tissue as a result of duct wall weakening and rupture. The main duct appears normal, but a definite decrease in the number of small duct radicals is noted.

The globular pattern is composed of larger accumulations of contrast solution, measuring 1 to 2 mm in diameter. Again, the major duct is normal but there is complete absence of the minor duct radicals. With the coalescence of globules, the cavitary pattern is formed. The areas of pooled, opaque solution are irregular in size, with a decrease in number but an increase in size of the globules. The last stage is referred to as destructive sialectasis. Bizarre patterns reflecting advanced lymphocytic infiltration and duct atrophy are seen, with the opaque solution dissecting its way into the residual gland because of loss and fragmentation of the duct walls.

The globular and destructive patterns may be confused with the "puddling" seen in sialographic studies of malignant disease of a salivary gland. Differentiation is frequently determined by the finding that more than one gland is involved in sialectasis, whereas neoplatic disease is almost always uniglanular.

As is expected, emptying of the sialographic solution is markedly delayed. Usually, it is retained indefinitely. The trapping of medium behind ductal obstructions and outside the limits of the duct lumen, in addition to inadequate salivary lavage, serves to retain the contrast solution. Therefore, when a diagnosis of antoimmune sialosis is suggested on the basis of clinical evidences, it is particularly important to select a sialographic medium that is more easily evacuated. Oil based solutions may promote the formation of foreign body granulomas and cause further gland destruction. In addition, media that retain their opacity for long periods will blot out future studies.

Treatment

Nusair S and Rubinow A (1999)[26] performed meta analysis of various studies from 1966 to 1998 in patients of primary SS and other inflammatory disorders. Oral pilocarpine was given in an optimal dose of 5 mg 4 times daily were less likely to cause side effects. A multi-center study in SS patients also suggests that oral pilocarpine is effective and safe for long-term administration. To combat dryness caused by SS or radiotherapy.

The therapeutic management has been quite disappointing. Steroid therapy may control glandular swelling, but no success has been obtained in curbing the inexorable progression of the disease process. Although irradiation may also control swelling, its implications must be considered. Therapy is usually symptomatic and may include salivary stimulation; duct probing, antibiotics, massage and therapeutic sialography, but these have not given uniformly satisfactory results.

Conservative surgical procedures in the form of major duct ligation or more radical operations, such as gland extirpation, have been employed for cosmetic purposes or when the gland is subjected to repeated and severe infections.

VIRAL INFECTION—MUMPS

This is caused by the Paramyxovirus and affects the salivary glands and the gonads/Central nervous system. Common between 5 and 10 years and comes in epidemics in schools and communities, easy to diagnose due to the painful swelling of the affected gland, low grade fever. When occurring in the adolescents care should be taken regards the Orchitis and epidedymitis. Meningitis and encephalitis are rare complications. Treatment is basically to control fever and pain. Tab crocin, 500 mg (Paracetomol) is the best.

Casella R et al (1997)[27] treated 11 patients with severe mumps orchitis. All patients showed marked scrotal swelling with a temperature above 38.5 C Serum C-reactive protein was significantly elevated (mean 140 mg./l.). None of the patients had been vaccinated. Antibodies to the mumps virus (IgG and IgM) were positive in all cases. The average interval between parotitis and onset of orchitis was 10 days. Treatment included bed rest with local cooling, scrotal support and systemic treatment with nonsteroidal anti-inflammatory drugs. Ciprofloxacin or clavulanic acid/amoxicillin should be administered if bacterial orchitis cannot be excluded.

Young adults Aspirin (Disprin,300 mg) 2 tabs tid is best. Good hydration and in small children iv drips may be indicated. These drips can be good vehicle for B complex group of vitamins in suitable doses. (Best in hospitalized conditions with attending pediatric dentist in attendance).

ACUTE SUPPURATIVE SIALADENITIS

This disease should be aggressively treated with Antibiotics. Suggested antibiotics are Vancomycin, combination of ampicillin and amoxycillin, are per doses recommended by the company inserts. Options for surgical drainage and surgical removal of the gland in cases of advanced destruction. It is best to do antibiotic sensitivity test in these cases prior to prescribing final regimen. Normally 7 to 14 days of antibiotics or more may be necessary (Fig. 18.3).

FIGURES 18.3A and B: 36-year-old female patient came with severe pain and fever of three days duration. Left angle of the mouth, pre-auricular region was tender and erythematous. The parotid duct on milking emanated with foul smelling pus. The pus was sent for culture and patient treated with combination of Amoxycillin and Cloxacillin (RedClox LB 500mg® for eleven days together with anti-inflammatory and analgesic preparation as required (Clinical diagnosis of Parotid Abscess was made, Girish Rao, Asha Iyengar, Nagesh KS, RV Dental College, Bangalore)

Wang S et al (1996)[29] have proposed a classification of chronic suppurative parotitis. It is suggested that CSP should be classified into recurrent parotitis in childhood (RPC), recurrent parotitis in adults (RPA), chronic obstructive parotitis (COP) and should be differentiated from other subdivisions including subclinical Sjögren's syndrome (SCSS), chronic parotid swelling of Sjögren's syndrome and sialadenosis with retrograde infection. RPA is a continuation of recurrent parotid swelling from childhood (RPC) to adulthood.

ALLERGIC SIALADENITIS

Here the history of ingestion of a particular drug, or foodstuff is elicited. Swelling of the lips bronchospasm and rarely anaphylactoid reactions may occur concomitantly. This has to be dealt like and emergency (See chapter on medical emergencies for Rx of allergic reactions).

SIALADENOSIS

This term (as used as Sialoses) actually describes a non malignant, non inflammatory enlargement of the salivary glands. Cause is unknown in most cases. Known to be associated with menopause, pregnancy, diabetes, alcoholism, Intestinal Surgery, Drug associared are phenylbutazone, iodine containing compounds etc. Salivary potassium elevations and sodium depletion is characteristically seen.

Kim D et al (1998)[31] have said that sialosis (sialadenosis) may be defined as an asymptomatic, non-inflammatory, non-neoplastic parenchymal salivary gland disease accompanied by a persistent painless bilateral swelling of the salivary glands, most commonly involving the parotid glands. There is no sex predilection, and the peak age incidence is between 30 and 70 years of age. Sialosis can occur due to three main causes **1**. alcoholism **2.** diabetes mellitus **3.** malnourishment. An autonomic neuropathy, seen as a demyelinating polyneuropathy, seems to be the common underlying basis.

SALIVARY GLAND NEOPLASMS

The vast majority of neoplasms are epithelial, originating from the secretory cells or ductal structures. The clinical courses of neoplastic diseases differs, depending for the most part of the cell type. And yet, with few exceptions, the clinical symptoms are essentially similar.

It has been adequately demonstrated that 80 percent of all salivary gland tumors occur in the parotid gland. Ten percent in the submandibular gland and the remainder in the sublingual and accessory salivary glands. It has also been shown that approximately 65 percent of all salivary gland tumors are benign and the remaining 35 percent malignant. However, the ratio of malignant to benign tumors is higher in the submandibular salivary glands than it is in the parotids.[4,5]

Except for the papillary cystadenoma lymphomatosum (Warthin's tumor), in which a 7:1 ratio favors the male, females are more likely to develop neoplastic diseases than

are males. Although the median age of tumor patients varies in accordance with the specific cell type, it is generally agreed that the fourth to seventh decades of life are the periods of greatest susceptibility.

In the parotid and submandibular salivary glands, the most frequent benign lesion is the benign mixed tumor. The most common malignant neoplasm of the parotid is the mucopidermoid carcinoma, followed by the malignant mixed tumor. The adenoid cystic carcinoma (cylindroma) is the most common submandibular malignant tumor.

Clinical Findings

Although neoplastic involvement is usually unilateral, bilateral lesions have been reported. The majority are unicentric in origin, but recurrent tumors are commonly believed to be multicentric.

Early in the development of a salivary gland neoplasm, when it is difficult to differentiate the benign from the malignant lesion, the patient may merely present with an asymptomatic, mobile enlargement. But as development continues, other clinical features appear which serve to distinguish the true nature of the mass. The benign salivary gland tumors are generally slow and steady in their growth, but a history of intermittent or rapid growth may occasionally be obtained. A more rapid rate of growth is usually suggestive of the malignant neoplasm. When faced with a mass that, according to the patient had been relatively static for many years but has suddenly undergone a period of rapid growth, thought must be given to the possibility that the mass is a malignant transformation of a benign tumor.

Wolf IS et al (1997)[33] have described the pleomorphic adenoma of the parotid to be the most common tumor of salivary gland origin, accounting for 60 to 70 percent of all benign salivary gland tumors. This lesion is usually a slow-growing painless mass inferior to the pinna of the ear. The diagnosis is based on clinical presentation and assited by fine-needle aspiration biopsy, magnetic resonance imaging or computed tomography.The treatment is wide excision in which the entire capsule is removed but the facial nerve is spared. Proper diagnosis and treatment are necessary to prevent the complications of tumor recurrence and malignant transformation. Carcinoma ex-pleomorphic adenoma arises in longstanding tumors and has a five-year recurrence rate of 75 percent.

Pain is commonly associated with malignant tumors of the salivary glands, but benign tumors may also lead to this complaint. In either case, the pain is a result of expansion within the gland's fibrous capsule that brings pressure upon the sensory nerves in the area. When the parotid gland is attacked by a malignant disease, involvement of the seventh nerve becomes evident in about 40 percent of such cases. The close intimacy of the facial nerve to the parotid parenchyma predisposes the nerve to infiltration by malignant cells. Weakness and/or complete paralysis of the facial musculature may ensue. The migration of the adenoid cystic carcinoma along nerve sheaths is unique and often is the cause of muscle paralysis and subjective pain.

Benign tumors of the parotid or submandibular salivary gland, when superficially located, may appear freely movable and circumscribed and have a soft to firm consistency. Benign mixed tumors because of their unique ability to form excrescences, often demonstrate clinically a bosselated configuration. Deeper, encapsulated masses may show a limited mobility and may not be as evident to palpation. The invasive characteristics of malignant tumors usually lead to fixation of the masses to surrounding tissues and on palpation these are generally found to be stony hard. Although the malignant mixed tumor also contains area of infiltration, this type is for the most part circumscribed by a capsule.

Lymphadenopathy, in the presence of a tumor, is an ominous sign. It must be assumed that the tumor is malignant and those metastases have occurred. However, the suspicion of malignancy in the absence of lymphadenopathy cannot be dismissed. At the initial examination, cervical node metastasis were found in only 15 percent of the patients with malignant mixed tumors, but positive node involvement was reported in 40 percent of the patients who returned with recurrences. Some additional aid in differential diagnosis may be obtained from the fact that benign tumors, particularly the mixed tumors and Warthin's tumor, usually involve the tail of the parotid. Primary malignant neoplasms are more frequently encountered in the superior portions of the gland.

Saku T et al (1997)[35] studied 145 malignant and benign tumors of the salivary glands. These were diagnosed during the period 1950-1987. Among 41 malignant tumors,

the frequency of mucoepidermoid tumor was disproportionately high at high radiation doses and among 94 benign tumors, the frequency of Warthin's tumor increased with increasing radiation dose. Their findings suggest a causal role for ionizing radiation in salivary gland tumor genesis.

Kusama K et al (1997)[36] analyzed 129 cases of minor salivary gland tumors diagnosed from 1970 to 1996. Eighty benign and 49 malignant minor salivary gland tumors were found in the approximately 9,300 oral biopsies submitted during the 27-year period. Pleomorphic adenomas were the most commonly histologic type of the benign tumors identified and 51 percent of the malignant tumors were diagnosed as mucoepidermoid carcinoma. Palate was the commonest location and there was a female predilection.

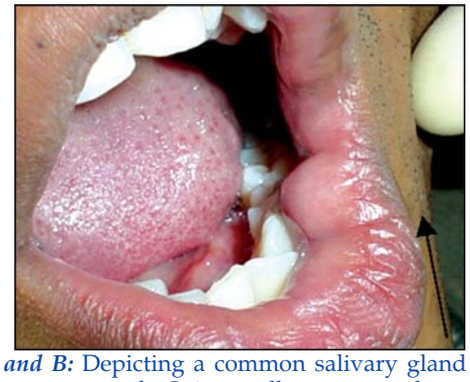
Ouoba K et al (1998)[34] have analyzed 48 cases of salivary gland tumors, composed of 28 women and 20 men with an average age of 41 years. Out of which 66.7% were from parotid, 20.8% were in the submandibular, and 12.5% were minor salivary gland tumors. Benign tumors were mostly pleomorphic adenomas (83.3%). The prognosis of malignant tumors with a survival rate of 20% in 3 years was unsatisfactory.

FIGURES 18.4A and B: Shows a raised bluish bullae on the buccal mucosa since five years. Histopathologically reported as Mucocele. (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College, Mangalore)

MUCOCELE

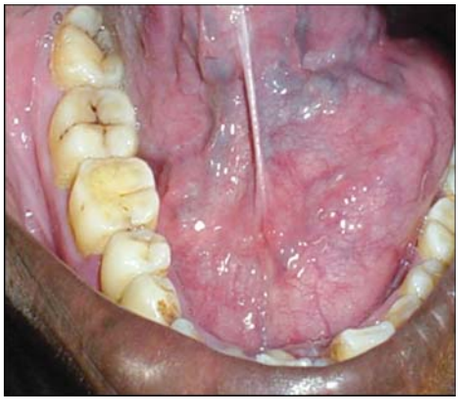
Mucocele is a term used to describe swelling caused by the pooling of saliva at the site of an injured minor salivary gland duct. The majority of these lesions occur on the lower lip. Mucoceles may be divided into a mucus extravasation type and a mucus-retention type. The mucus-extravasation type is the common mucocele and is caused by the laceration of a minor salivary gland duct by trauma. Saliva leaks into the submucosal tissues causing pooling of mucus, resulting in inflammation and formation of granulation tissue. The mucus-retention type is less common and is caused by obstruction of a minor salivary gland duct which causes a back-up of saliva. This continual pressure dilates the duct and forms a cyst like lesion.

Clinically they appear as Bluish raised thin walled lesion, when they are superficial, the deeper variety is soft fluctuate covered with normal oral mucosa which remains



FIGURES 18.5A and B: Depicting a common salivary gland mucus retention cyst, mucocele. It is usually seen as a dome shaped swelling on the occlusal line and in the lower lip (Nillofer S, Prasanna K, Bailoor DN 2003 Yenepoya Dental College, Mangalore)

for a long time till surgically removed (Figs 18.4 and 18.5). Ranula is special type of mucocele which grows in the floor of the mouth, unilateral and is called so due to its resemblance to the belly of the frog (Fig. 18.6).



FIGURES 18.6A and B: Figure shows extra and intraoral photographs of a female patient with plunging Ranula (Prasanna K, Nillofer S, Bailoor DN 2003 Yenepoya Dental College, Mangalore)

Treatment

Wilcox et al (1978)[1] suggest injection of corticosteroids before the surgery is attempted. The treatment of deep mucoceles or recurring superficial mucoceles is surgical removal of the lesion. A problem in management is that surgery to remove mucoceles may be responsible for the formation of new lesions by causing trauma to other minor salivary gland ducts. Large ranulas are often successfully managed by marsupialization rather than surgical removal.

DEVELOPMENTAL ANOMALIES

Aberrant Salivary Glands

An aberrant (or ectopic) salivary gland is salivary gland tissue that develops at a site where it is not normally found.

Ectopic glands are reported as a single anomaly or in combination with other facial anomalies. Sinha[3] described a patient with multiple anomalies including unilateral tonsillar aplasias, absence of a normal external auditory meatus and an ectopic salivary gland in the tongue. Most aberrant salivary glands of the neck occur in the upper portion of the neck.

Aplasia and Hypoplasia

Total aplasia of the major salivary glands is rare.

Hypoplasia of the parotid glands has been reported to be present in patients with Melkerson-Rosenthal syndrome, which chiefly consists of facial paralysis, facial edema and fissured tongue.

Accessory Ducts

Accessory parotid ducts are common. Rauch[4] studied 450 salivary glands and found an accessory parotid duct in over one half of the cases. This accessory duct was most frequently found superior and anterior to the normal Stensen's duct orifice.

Diverticuli

Diverticuli are small pouches or outpocketings of the ductal system of one of the major salivary glands and their presence leads to repeated episodes of acute parotitis. Diagnosis is made by a sialogram.

SUMMARY

Salivary gland disease is the clinical area in which all the doctors, the ENT, the Oral surgeons, Oral Medicine and Radiology, General medicine and family practitioners all need to work together so that patient gets the maximum benefit from their expertise.

All dentists must have a sound knowledge of the pathogenesis of the salivary gland diseases and what

labels are applied to each diagnostically. In testing, the non-invasive diagnostic procedures must be preferred over invasive ones, and in general the treatment should be chosen such as not to portray a knife happy surgeon. How the saliva or lack of it can affect the dynamics of the oral ecosystem should be clearly understood.

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Bailoor DN, Karthikeya Patil,
Mahima Patil, Varghese Mani

19 Odontogenic Tumors

INTRODUCTION

Odontogenic tumors (OT) are lesions derived from cellular elements that are forming the tooth apparatus. As a general rule this group of lesions do not cause pain and hence are neglected for a long time. The expansion of mandible or maxilla may result in facial deformity and swelling which brings the patient to the dentist.

Depending on which stage of the tooth development the pathological processes strike decides how the end lesion turns out to be. For example if in the bud stage itself the neoplastic changes strike the cells then chance of this lesion turning into Ameloblastoma (AMB) or any of its variants is very high. However, if the changes strike the advanced bell stage where the hard tissues of Enamel and dentin are being laid down then it is possible that odontoma or its variants may result (Fig. 19.1).

Another important criteria is the type of change that may affect the cell undergoing the tooth building process, whether it is neoplastic, hamartomatous or cystic.

Philipsen HP et al (1997)[10] has suggested a plausible hypothesis in which they mention that the pathogenesis of this group of tumors can take two paths. The neoplastic path and the hamartomatous path. The neoplastic path would lead to the Ameloblastic Fibroma and the hamartomatous path to the Odontoma group of lesions.

The authors would like to add that the cystic processes are the third type of forces of pathogenesis that may

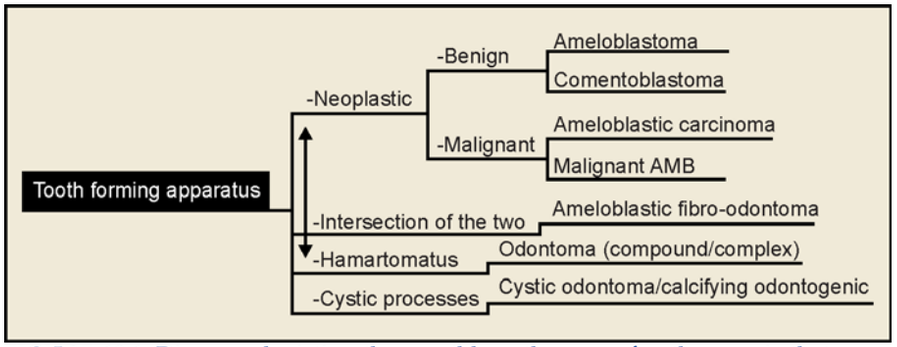


FIGURE 19.1: Diagram depicting the possible pathways of pathogenesis that may result in the wide variation of lesions observed arising from the remnants of the odontogenic epithelium and mesenchyme or its interaction

predominate in the diagnosis of Calcifying Odontogenic Cyst or the Cystic odontome.

Mosqueda-Taylor A et al (1997)[14] analyzed 349 odontogenic tumors and found 99% were benign and 1% malignant in their series. The most frequently occurring tumors were Odontoma (34.6%), Ameloblastoma (23.7%), Myxoma (17.7%), Adenomatoid odontogenic tumor (7.1%), and Calcifying odontogenic cyst (6.8%). Amongst the malignant they found 3 were PIC (Primary intra-osseous Carcinoma) and 1 was Malignant AMB (Fig. 19.2).

For our practicing dentist we present a simple classification, which will also serve the needs of proper diagnostic labeling or treatment selection.

EPITHELIAL TUMORS

a. Ameloblastoma (AMB)
b. Calcifying epithelial odontogenic tumor (CEOT)
c. Adenomatoid odontogenic tumor (AOT)
d. Rare variance—Clear cell Odontogenic tumor (CCOT), Squamous odontogenic tumor (SOT)—These will not be discussed since their rarity does not allow their discussion.

Ameloblastoma (AMB)

This neoplasm originates in cell rests of Malassez, cell rests of serrae, other areas of reduced enamel epithelium and epithelial lining of cysts like Dentigerous cyst. Chidzonga MM et al (1996)[17] surveyed 1,723 biopsies in a ten year time period in Zimbabwe and determined that the commonest non-odontogenic tumor was epidermoid carcinoma, commonest cyst was Dentigerous cyst and the Ameloblastoma constituted 79.1% of the odontogenic lesions, in their series.

Clinical Features

Equal distribution in males and females with age ranging from 35 to 45 years. Ameloblastoma may occur anywhere in the jaws. The most frequent site appears to be molar ramus region (Fig. 19.6).

Gardner DG (1996)[1] mentions about the three clinical types of AMB I. Solid (Muticystic-Classic) II. Unicystic III. Peripheral variety. In his opinion it is the structure of the cancellous and the compact bone in the region of initiation of the AMB which ultimately decides how it will spread and radiographically what kind of picture it will present.

Parate SN et al (1999)[2] have highlighted on a case of malignant Ameloblastoma (AMB) which they were able to diagnose on basis of a combination of Fine Needle aspiration Biopsy (FNAB) and regular Histopathological examination. They also caution on the limitations of the FNAB specially when it comes out negative. In such cases the clinician must go by his gut feeling and experience and choose to do further diagnostics. The term malignant Ameloblastoma is reserved for those metastasizing or fast growing locally destructive tumors that retain the typical morphology of AMB.

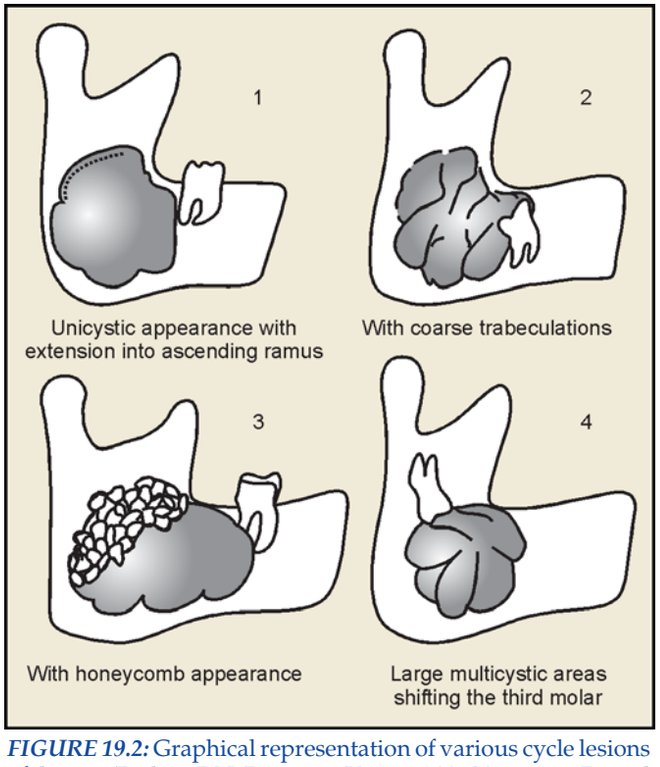
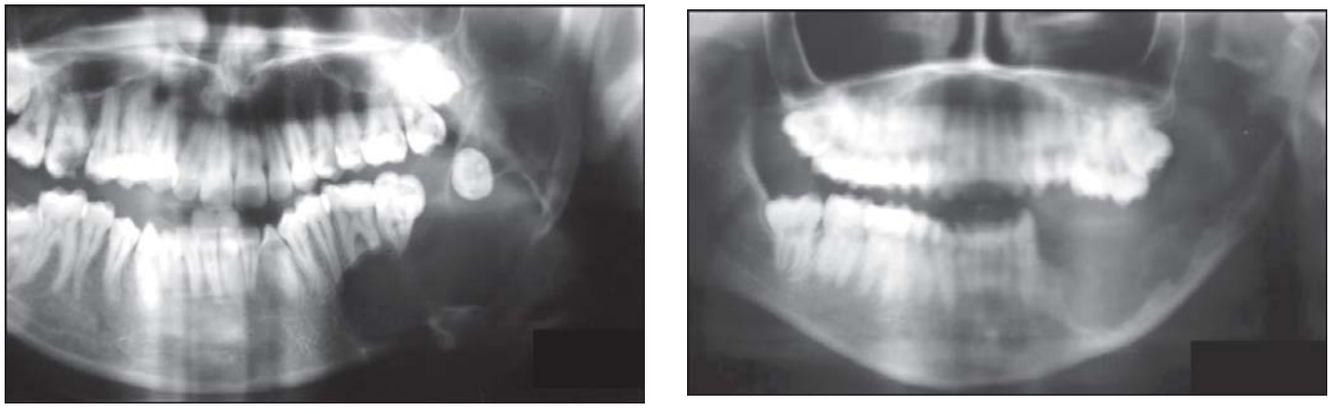


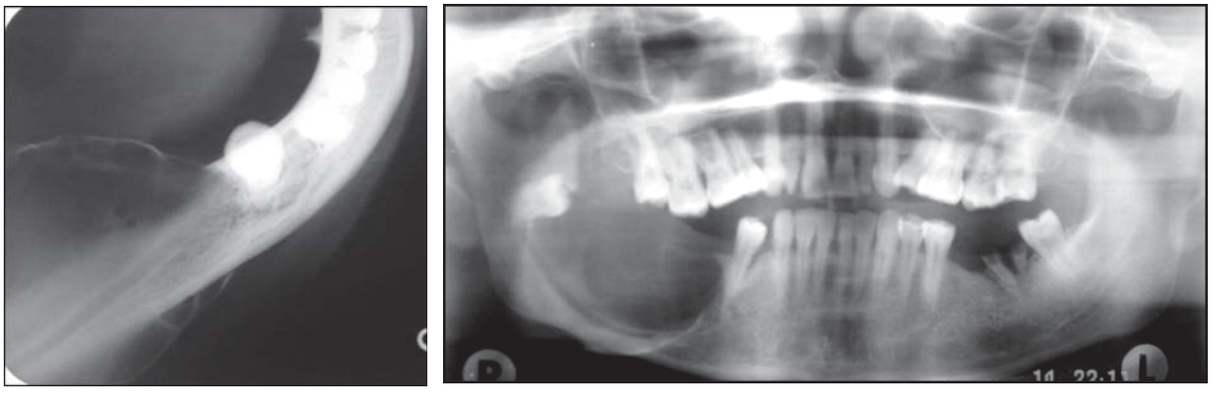
FIGURE 19.2: Graphical representation of various cycle lesions of the jaw (Bailoor DN, Prasanna Kumar 2004 Yenepoya Dental College, Mangalore)

Bommer KK et al (1997) [3] in a classical study evaluating the Fine-needle aspiration (FNA) to distinguish neoplastic versus non-neoplastic lesions in many organs studied the cytology of 450 bone lesions. The spine was the most frequently aspirated site (49%), followed by the ilium, sacrum, mandible, ribs, and femur. Three hundred and eighty-five aspirates (86%) were adequate for evaluation, with 215 cases diagnosed cytologically as positive for

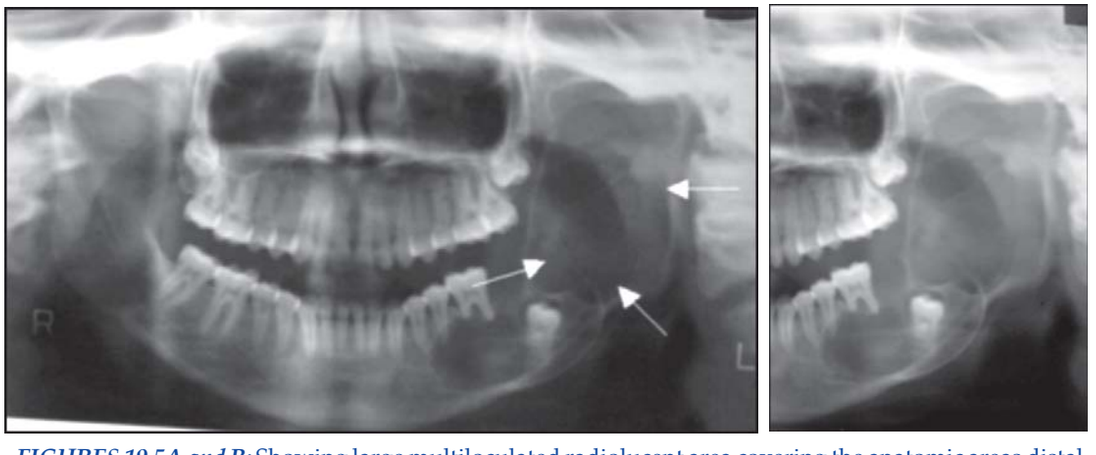


PREOPERATIVE *POSTOPERATIVE*

FIGURES 19.3A and B: Shows large radiolucent destruction of body of mandible and ascending ramus. The teeth in proximity show root resorption. Histopathologically confirmed to be ameloblastoma. It is pre-operative and post-operative as suggested



FIGURES 19.4A and B: Shows unilocular radiolucent lesion showing lingual expansion and light trabaculation. More destruction of ascending ramus and loss of superior border of mandible has been described by Worth HM as classical for ameloblastoma (Courtesy: Varghese Mani GDC Calicut 2004)



FIGURES 19.5A and B: Showing large multiloculated radiolucent area covering the anatomic areas distal to first Mandibular premolar to the condylar region including the ascending ramus. The molar tooth appears to be embedded in the lesion. Histopathologically Ameloblastoma was reported (Keerthilatha Pai 2004 College of Dental Surgery, KMC, Manipal 576 119)

malignancy, 11 cases as suspicious but not diagnostic of malignancy, and 2 cases as inconclusive Metastatic carcinoma was present in 175 of the 215 malignant aspirates, and 67 percent of these were adenocarcinomas.

FNA biopsy of bone lesions is a reliable and easily performed diagnostic test for metastatic and primary bone tumors. The simplicity and accuracy of this procedure, which does not require any surgical incisions (open biopsy or manipulation), supports its important role in managing bone lesions with minimum risk or morbidity.

Radiographic Appearance

Radiographically four variations of Ameloblastoma are seen in the clinics

a. Unilocular cyst like radiolucency in ascending ramus region with destruction of anterior part of coronoid (Fig. 19.3).
b. Multilocular radiolucency with coarse trabeculations (Fig. 19.6).
c. Multilocular radiolucency with fine honeycomb of trabeculation.
d. Large angular compartments of radiolucent areas within a single large loculated radiolucency (Figs 19.4 and 19.5).

Kawai T et al (1998)[4] reported the findings on conventional radiography including panoramic, postero-anterior and Waters' projection, and the findings of computed tomography (CT) and magnetic resonance (MR) imaging. These were evaluated using the following three variables: artifact *degradation, lesion detectability, and conspicuity* (Figs 19.7 and 19.8). The results suggested that MR imaging was the best imaging method for visualization of the tumors, followed by contrast enhanced CT.

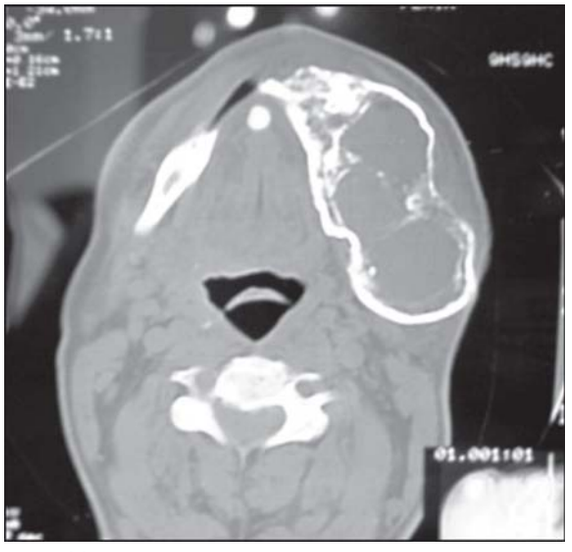
Histopathology

There are many histologic patterns described and standardized for serious research. However, for a practising dentist it is enough to know that it is a variant of Ameloblastoma and for surgeon to plan a total surgical resection.

All the subtypes of this order appear as a pattern of loosely arranged cells that mimic stellate reticulum of the enamel organ. Sometimes the cystic degeneration of the follicular islands leads to the diagnosis of cystic Ameloblastoma.

Weir MM et al (1998)[5] describe an unusual case of malignant (metastatic) ameloblastoma with histological confirmation. Characteristic cytological findings included

FIGURES 19.6A to D: Shows extra and intraoral photographs of 37-year-old male patient with diffuse swelling in lower right third of the face. Radiograph shows multilocular radiolucency (soap bubble). Histologically reported as Ameloblastoma (Gopakumar N, 2004, AB Shetty Memorial Institute of Dental Sciences, Mangalore 575018)



FIGURES 19.7A and B: A 43-year-old male with non painful swelling of four years duration. CT scan revealed that expansion of both lingual and buccal aspects and the coarse trabeculation and multilocular images. Histopathology confirmed Ameloblastoma (Borle Rajiv et al, Sharad Pawar Dental College, Wardha 2003)

fibrovascular central cores surrounded by palisading crowded basaloid or columnar cells or both and rosette-like structures of tumor cells with central fibrillary material.

Follow-up of AMB

Hayashi N et al (1997)[6] mention a case of Ameloblastoma of the mandible, which metastasized to the orbit with malignant transformation. A 63-year-old woman who was surgically treated for mandibular Ameloblastoma 27 years previously, had the tumor which recurred three times in the past 5 years. The orbital tumor and recurrent ameloblastomas were investigated histopathologically and histochemically. The tumor changed in morphology as it recurred, from follicular ameloblastoma without atypia to apparent malignant tumors disclosing undifferentiated or squamoid features. This further emphasizes the fact that after surgical excision it is always better to do a long term follow up of all the cases.

Differential Diagnosis (DD)

The following must be considered in the DD.

- CEOT
- Odontogenic keratocyst (OKC)
- Dentigerous cyst
- Odontogenic myxoma
- Giant cell lesion
- Ossifying fibroma
- Central hemangioma.

Treatment

Total surgical resection is the treatment of choice.

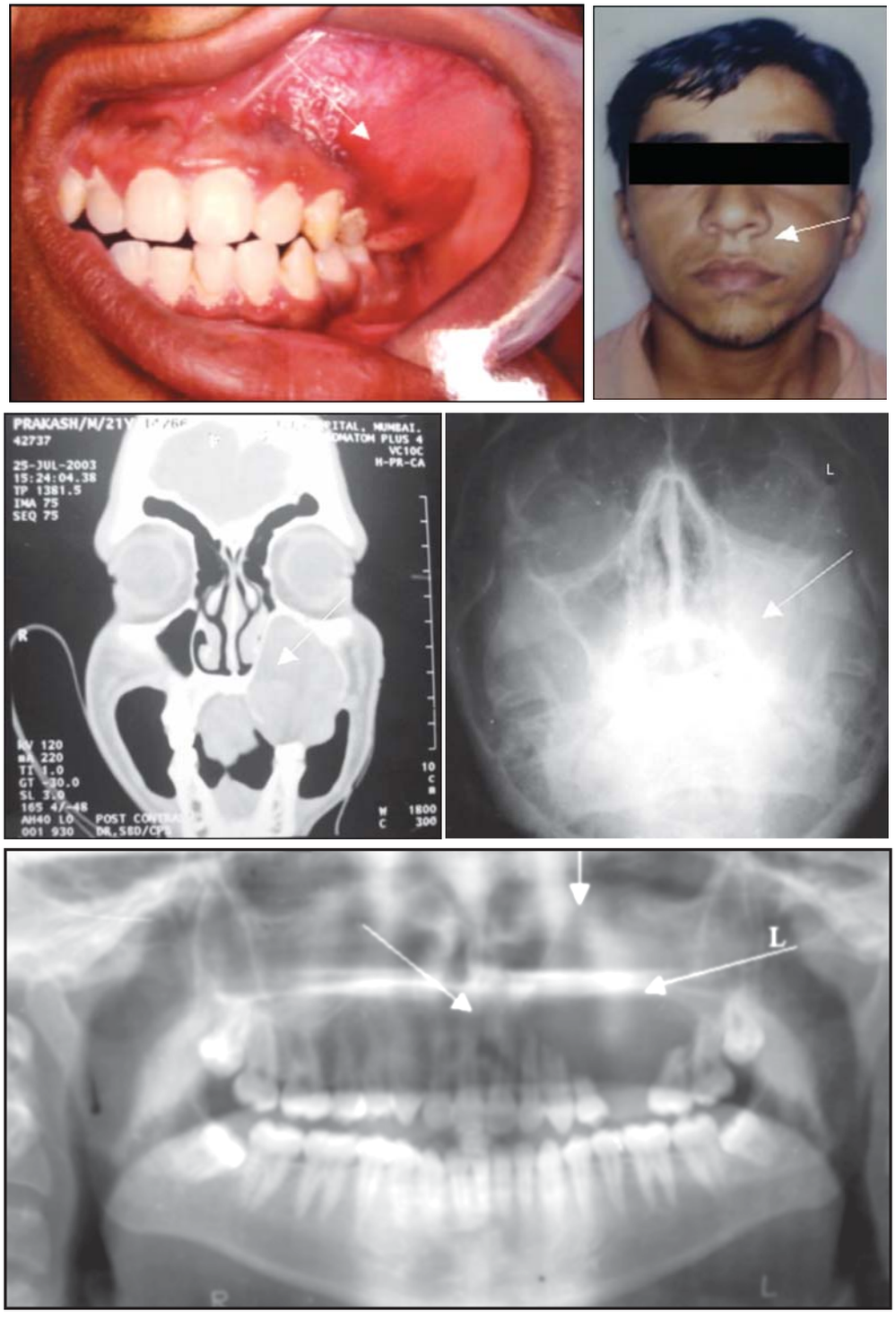
Calcifying Epithelial Odontogenic Tumor (CEOT)

This tumor has been traditionally called as Pindborg's tumor in reference to the pathologist who described this lesion for the first time.

Ng KH and Siar CH (1996)[7] reviewed the clinicopathological characteristics of 13 cases of calcifying epithelial odontogenic tumor (CEOT) (Pindborg tumor) diagnosed in the Institute for Medical Research, Kuala Lumpur, over a 29-year period. These consisted of eight (61.5 %) Malays, three (23.1 %) Chinese, one (7.7 %) Indian and one (7.7 %) Melanau. Their ages at presentation ranged from 19 to 61 years (mean age, 31.8 years). There were 12 central and one peripheral CEOT. Around 75 percent of the tumors were located in maxilla. In more than 50 percent of the cases clinical diagnosis was made as dentigerous cyst.

Clinical Features

There seems to be no gender predisposition and the mean age is 40 years similar to Ameloblastoma. Some series show mandible lesion frequency to be twice that of maxilla



FIGURES 19.8A to E: Showing a sixteen-year-old male came with complaint of painless swelling of the left molar region since last four years. Radiolucent lesion of the maxillary sinus appeared to caused the resorbtion of the canine, premolar and molar teeth. Histopathological diagnosis of Ameloblastoma was made (Ani John, Hemant Umarji GDC Mumbai 2004)

and in mandible molar ramus region seems to be favored. Painless expansion of facial bones is the only early feature in most cases.

Radiographic Features

CEOT is frequently associated with unerupted teeth. Radiographically, lesions may be radiolucent unilocular and sometimes multilocular. This radiolucent area more often than not, contains radiopaque peppering of fine particles which have been described to be very characteristic. (*Driven snow appearance*)

Histopathological Features

CEOT has reasonably typical appearance. Sheets of large polygonal epithelial cells with nuclei showing large variation in size, shape and number. The cytoplasm is eosinophilic in color. Differing ratio of amyloid is seen typically in extracellular areas. This pale staining eosinophilic material stains positive with Congo red stain. Negri P et al (1999)[8] mentioned about the histologic features revealed the presence of a homogeneous substance resembling amyloid and many cells filled by calcified material in the form of concentric Liesegang's rings.

Differential Diagnosis

The following lesions should be differentiated from this CEOT due to close clinical radiographic or histopathological features.

1. Dentigerous cyst
2. Odontogenic kerotocyst (OKC)
3. Ameloblastoma
4. Odontogenic myxoma
5. Adenematoid odontogenic tumor (AOT)
6. Ossifying fibroma

Treatment

Surgical enucleation to total resection seems to be the options usually available. Metastases or malignant conversion are rare.

Adenomatoid Odontogenic Tumor (AOT)

This is believed to be a Hamartoma rather than a neoplasm.

Clinical Features

Age range of 6 to 30 years is seen in most series. Females are affected more. Most lesions appeared in anterior portion of the jaws. It is often associated with the crowns of unerupted teeth. It has a well-circumscribed capsule.

Radiographically

Unilocular Radiolucent well circumscribed lesion around the tooth well within the jaws. Some of them have a peppering of radiopaque spots throughout the radiolucency. This probably is due to the pre-enamel tissue in various stages of calcification (Fig. 19.9).

FIGURES 19.9A and B: A 16-year-old patient presented with painless swelling of the left maxillary buccal region. Radiograph revealed clearly demarcated radiopaque lesion with peppering of radiopacity (Gopakumar et al AB Shetty Memorial Institute of Dental Sciences, Mangalore)

Histopathology

Duct like structure of columnar epithelial cells are seen in this lesion. This is seen together with the epithelial proliferation of polyhydral to spindle shaped cells. Many points of enamel like material is seen interspersed throughout the tumor.

Differential Diagnosis

AOT needs to be differentiated with

1. Dentigerous cyst
2. Lateral root cyst
3. Calcifying odontogenic cyst
4. CEOT

Treatment

Enucleation is sufficient in most cases and recurrence is rare.

TUMORS ARISING FROM MESENCHYMAL ODONTOGENIC TISSUES

a. Odontogenic myxoma
b. Central odontogenic fibroma
c. Cementifying fibroma
d. Cementoblastoma (True cementoma)
e. Periapical cemental dysplasia

Odontogenic Myxoma

This tumor consists primarily of follicular connective tissue appearing histopathologically close to the structure of the pulp. When the proportion of collagen in the tissue is more it is likely to be given a label of 'Myxo-fibroma by the histopathologist. Recurrence has been noted in quite a few cases.

Clinical Features

No gender predisposition is noted, with equal frequency in both the jaws and the age range of the patients is given to be between 10 and 50 years, frequency peaking at 30 years.

Radiographic Features

Well circumscribed radiolucent lesion, sometimes showing a multilocular and honeycombed pattern. Cortical expansion and root displacement is usually observed on the radiographs.

Histopathological Features

In a matrix of mucopolysacchrides a relatively sparse spread of fibroblasts, myofibroblasts and collagen fibrils are usually found. Islands of bony tissue with capillaries representing the bony trabeculae of the honeycombed appearance is seen in the radiographs.

Differential Diagnosis

Following lesions are to be differentiated.

1. Ameloblastoma
2. Central hemangioma
3. Normal developing tooth bud

Treatment

Complete surgical excision is the key to prevent the recurrence. Even in cases where recurrence is seen no metastasis has been noted.

Central Odontogenic Fibroma

It is a rare tumor which presents *radiographically* as radiolucent lesion which is multilocular with cortical expansion. No age and jaw predilection is mentioned. *Histopathologically* two types of tumors have been described (i) Simple type in which a mass of mature fibrous tissue containing few epithelial rests. (ii) In the WHO type mature connective tissue is superadded with calcific rests which may be identified as dentin or cementum. No clinical difference has been noted between the two histopathological types.

Cementifying Fibroma

This lesion presents as one of the variants of the central fibrous lesions of the jaws. Histopathologically it may be difficult to distinguish between the cementum and the immature bone tissue and other calcified material (Fig. 19.10).

Histopathological Features

Cementum is usually identified as globules or oval islands of calcified material in a fibrous stroma. These oval areas may be surrounded eosinophilic immature cementum

tissue and cementoblasts. Gradually maturing tumors have increasing areas of globular cemental masses and lesser of the fibrous stroma.

FIGURE 19.10: A thirty nine year old female with bony hard slow growing swelling. Histopathological report revealed cementifying fibroma (Borle Rajiv et al, Sharad Pawar Dental College, Wardha, Maharashtra)

Differential Diagnosis

Following are the lesions that need to be considered in the DD:

1. Cementoblastoma
2. Ossifying fibroma
3. Chronic osteomyelitis
4. Fibrous dysplasia
5. Ameloblastoma
6. Odontogenic myxoma

Treatment

Well circumscribed nature and slow growth of this lesion makes it ideal candidate for conservative surgical approach since there is no danger of recurrence.

Cementoblastoma

It is a benign tumor of the cementoblasts and is often termed as the 'true' Cementoma. Highest frequency is reported in the second and the third decade of life. Equal in both sexes, and more commonly seen in mandible than in maxilla. It is related to the periapical region and connected with the roots of the teeth involved. Tooth remains vital. Cortical expansion is seen and in many patients low-grade pain has been reported intermittently.

Radiographic Features

It is a radiopaque lesion attached to the root surrounded by a radiolucent ring.

Histopathologic Features

An assembly of cementoid tissue with irregular reversal lines. Vascular areas with hyperchromatic cementoblasts and cementoclasts interspersed with each other.

Differential Diagnosis

This lesion needs to be differentiated from

1. Odontoma
2. Osteoblastoma
3. Focal sclerosing osteomyelitis
4. Hypercementosis

Treatment

Unless severe pain or facial deformity intervenes, a very conservative approach is favoured by most clinicians. If the lesion has to be removed then the tooth also has to be sacrificed.

Periapical Cemental Dysplasia

This diagnostic label is more suggestive of a reactive lesion rather than a neoplastic one. Trauma and infection may cause the unusual reaction in the bone and cemental tissue. However, debate continues as to the true nature of this lesion.

Clinical Features

This condition is seen more in the black race, more in women and most in the mandibular anterior region. It is common to see more than one teeth involved at one time. Most teeth are without any symptoms and routine radiography is the reason why these lesions are discovered. All the teeth associated with PCD are vital.

Radiographic Features

It appears as multiple or single radiolucency in relation to the tooth tip. The radiolucency is continuous with the periodontal ligament space. Gradually it may be peppered with radiopacity and whorled patterns in the radiolucency

and in period ranging from 2 to 4 years a solid radiopaque mass with a thin margin of radiolucency is evident. Different teeth adjacent to each other may show differing stages of maturation.

Florid Osseous Dysplasia (FOD)

FOD is a rare and fast growing form of this condition. It is seen bilaterally affecting both the jaws. Some investigators have reported the presence of the traumatic bone cyst associated with this condition. FOD needs to be differentiated from Sclerosing osteomyelitis, and Paget's disease.

Untreated FOD can convert into osteomyelitis or case of malignancy has also been reported. Schneider LC et al (1999)[13] have reported a case of 54-year-old female of African origin whose biopsy report came as spindle cell malignancy of the mandible, after she developed severe pain and discomfort in the existing FOD lesions which were long standing. Patient died in 20 months despite the best efforts of surgeons and radiotherapists.

Histopathological Features

This condition is a mixture of fibrous tissue, bone both mature and immature and cemental tissue. The calcified tissues are usually arranged in spicules and irregular masses. Reversal lines are seen in the calcified tissues. It is many times very difficult to distinguish between the periapical fibroma and this condition. If the inflammatory cells predominate then it may mimic osteomyelitis.

Differential Diagnosis (DD)

Following conditions

1. Periapical cyst
2. Periapical granuloma
3. Periapical abscess
4. Ossifying fibroma
5. Chronic osteomyelitis should be clinically differentiated.

In the opaque stages the DD would be odontoma, osteoblastoma, and focal sclerosing osteomyelitis.

Treatment

Usually no treatment is necessary except when the lesion is aggressive and may convert to FOD.

MIXED—EPITHELIAL AND MESENCHYMAL TUMORS

- Odontoma
- Ameloblastic Fibroma/Ameloblastic Fibro-odontoma

Odontoma

They are most common odontogenic tumors. Both the epithelial and mesenchymal origin tissue go into the formation of this benign neoplasm. Most of the investigators today believe that odontomas contain the mature enamel and dentinal tissue with a disturbed orientation and architecture and therefore, qualify to be termed truly as 'hamartomas'.

Depending on the level of the disturbance the radiographs may reveal either a bunch of little teeth or teeth like structures in focal powdered radiopacity (*Compound odontoma*) (Figs 19.11 and 19.12), or it may have broken down into fine powder like amorphous material of various sizes aggregated giving it a mixed radiopaque —radiolucent appearance (*Complex odontoma*) (Figs 19.13 and 19.14).

Ng KH and Siar CH[12] 1997 have reviewed the clinicopathologic characteristics of 104 cases of odontoma over a 29-year period (1967-1995). The results showed no real predilection in terms of sex, race or site distribution. The mean age at presentation was 24.8. There were 102 intraosseous and 2 extraosseous odontomas. Swelling was the most common presenting complaint. Compound (43.3%) and complex (35.5%) odontoma were the two most common histological types encountered.

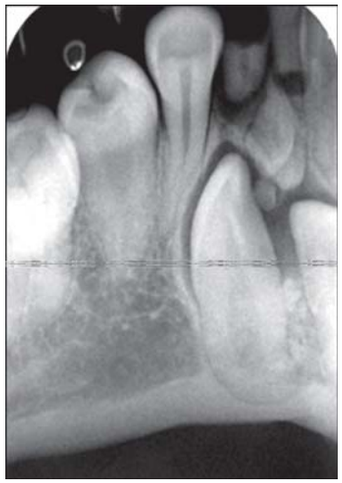
Clinical Features

Most cases are detected in children and adolescents in which weird tooth like substances may be seen erupting or discovered on routine radiography. In most cases these lesions are asymptomatic. Gender and jaw predilections are not very strong. Compound odontoma have been observed more in the anterior region in some series. Alveolar swelling is the only finding in many patients.

Radiographic Features

The lesions appear in the tooth bearing areas of mandible and maxilla. The compound odontoma has many small

FIGURES 19.11A and B: Depicting the occlusal and IOPA radiographs of patient with compound odontome, which has hindered the eruption of the canine (Omal PM, Beena K, Bailoor DN 2003 Yenepoya Dental College and Hospital, Mangalore)



FIGURES 19.12A and B: Depicting multiple compound odontomes in the periapical area of lower central incisor (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

tooth like radiopacities and the complex odontoma has a radiolucent area with a radiopaque border inter-spread with powdery radiopaque appearance. Sometimes a large radiolucent area pre-dominates in with this appearance and the radiologist may give a radiographic diagnosis of cystic odontome.

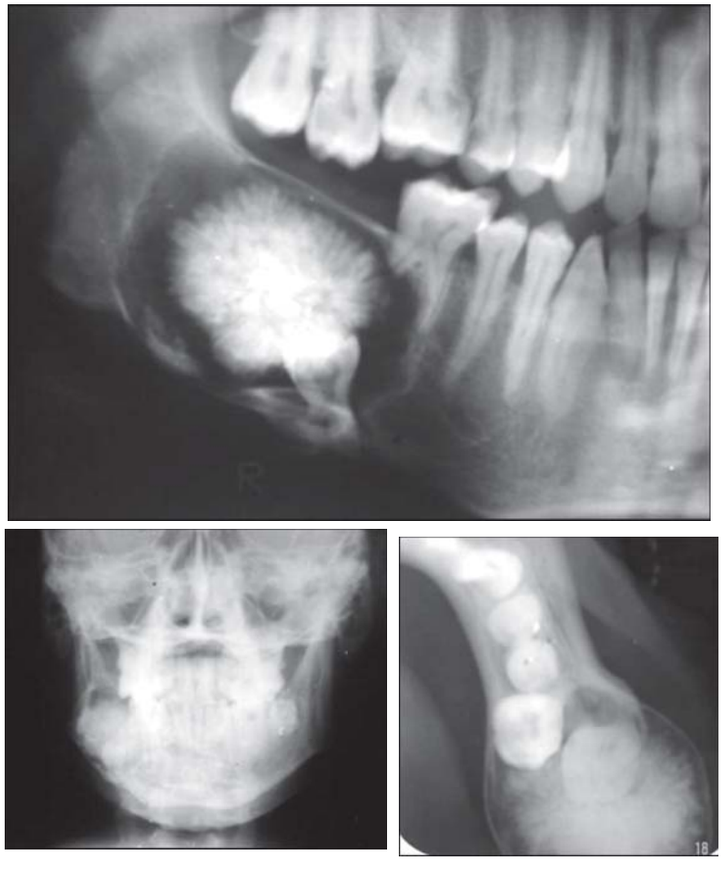
Histopathological Features

As may be expected the tissues of enamel, dentin and pulp may be observed in various levels of differentiation and in the complex type the resemblance to normal tissue may be difficult to see very easily. Ghost cell keratinizatin has been mentioned in some histopathological reports but its significance is not clear.

Differential Diagnosis

These lesions need to be differentiated from:

1. Focal sclerosing osteomylitis
2. Osteoma
3. Periapical cemental dysplasia



FIGURES 19.13A to C: Figure showing a 20-year-old male with bony hard painless swelling. OPG, PA skull and occlusal radiograph confirmed an radiopaque mass surrounded by radiolucent halo blocking the erupting second mandibular molar (Ani John, Hemant Umarji GDC Mumbai 2004)

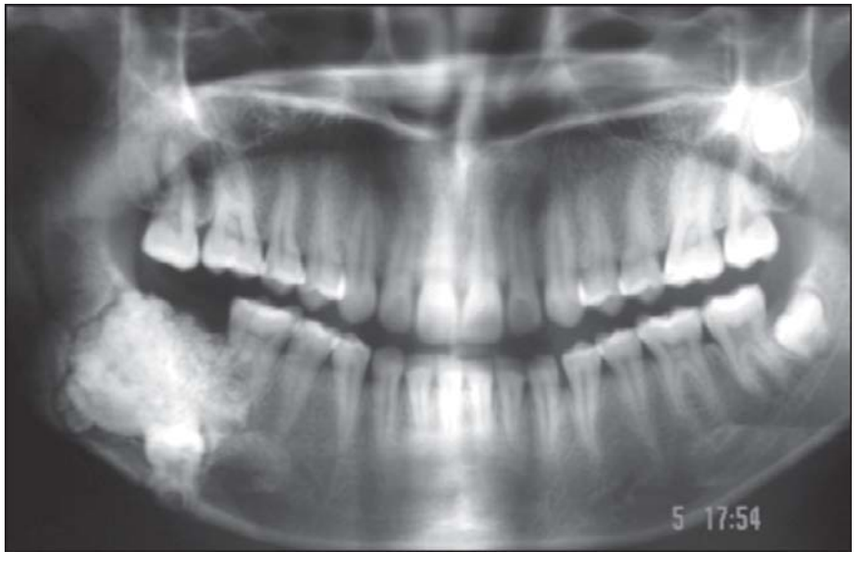


FIGURE 19.14: A 15-year-old patient complained of white irregular mass protruding from the gums in the right mandibular region. OPG revealed radiopaque mass blocking the eruption of the third molar- Odontome (Rajiv Borle et al, Sharad Pawar Dental College, Wardha, Maharashtra)

4. Ossifying fibroma
5. Cementoblastoma.

Treatment

Many of the odontomas need to be enucleated since they block the teeth from erupting or erupt in place of a permanent tooth.

Some of the odontomas may undergo secondary infection. Di Tommaso L et al (1998)[11] have mentioned a case in which Actinomyces infection was noted in a patient with compound odontoma in normal immune patient. This suggests that it may be possible that such lesions may act as nidus for infection and need to be promptly treated. Only in rare cases do they cause enlargement of the alveolar bone and may have predominant cystic elements.

Ameloblastic Fibroma (AF)/Ameloblastic Fibro-odontoma (AFO)

This neoplasm is seen in the young adults up to 20 years of age. Mandibular molar and ramus area seems to have the highest frequency of these lesions. Apart from a very characteristic radiographic and histopathologic appearance the cortical bone expansion is large and can cause deformity.

Ameloblastic fibro-odontoma occurs predominantly in children and young adults. The mandibular molar-ramus area is the favored location and radiographically, these lesions are well circumscribed and lucent-opaque. The tumor mass is composed of a myxoid connective tissue with strands of odontogenic epithelium and differentiated tissues such as enamel and dentin. The treatment is a conservative surgical procedure due to benign biological behavior. Ozer E et al (1997)[15] presented a 7-year-old girl presenting with proptosis. A partially ossified mass occupying the right maxillary sinus was found. A complete enucleation was performed after the histological diagnosis of ameloblastic fibro-odontoma

Owens BM et al [9] in a retrospective study of 104 cases of odontomas found that the vast majority were compound odontomas (64.4%) with complex odontoma comprising 31.0% of the total lesions. No ameloblastic fibro-odontoma were diagnosed in this series attritbuting to their rarity. In their series male predominance and maxillary lesions constituted 85% of the total. A maximum number of these tumors were associated clinically with over-retained deciduous teeth and un-erupted permanent teeth.

Ameloblastic fibrosarcoma has been documented but it is unclear whether is arises as a separate lesion or converts from the benign one. The rarity of this lesion precludes dogmatic assertions about this.

Radiographic Features

Well circumscribed, radiolucent with sclerotic margins. They may be unilocular or multilocular with crown of impacted teeth evident in them. A discrete area of radiopaque region representing the odontoma can be usually seen seperately, rarely totally chaotic radiopaque/radiolucent lesions with cortical expansion is seen on occlusal or PNS radiographs.

Histopathological Features

There is a definitive fibrous capsule. The stroma is consisting of primitive myxoid connective tissue. In between this stroma is seen two cell wide epithelium which has similarity to dental lamina which proliferates from oral epithelium. Some of these epithelial foci proceed and make enamel and dentin in abnormal shapes leading to the diagnostic label of Ameloblastic fibro-odontoma. Many investigator believe that these two entities are actually the two stages of the same benign neoplastic process affecting the jaws.

Differential Diagnosis

The lesions which can look close, radiographically or histopathologically are:

1. Ameloblastoma
2. Odontogenic myxoma
3. Dentigerous cyst
4. Odontogenic keratocyst
5. Central giant cell granuloma
6. Histiocytosis–X group of lesions.

Radiographically mixed patterns are also seen in CEOT, COC, Odontoma, and AOT.

Treatment

Conservative surgical procedure such as curettage and enucleation is usually enough since it is a well encapsulated lesion. Recurrence is rare.

CONCLUSION

Most of the benign tumors require radiographic and histopathological reports to be in hand before the Maxillo-facial surgeons can go in remove the lesion. Surgical treatment is a must in most cases of these pathologies and rates of recurrence must be kept in mind prior to planning the type of surgery and follow up.

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20 Reactive Lesions and Nonodontogenic Tumors

INTRODUCTION

A wide gamut of the swellings and tumors of the non-odontogenic origin have been included in this chapter. Some are just lesions originating from the minor irritations of the oral cavity and they have been included in the beginning of the chapter due to their relatively common occurrence. In the other groups most are self-limiting and come under the benign banner and the really wicked ones, the malignant groups are those that cause wanton destruction of tissues and need very aggressive treatment.

REACTIVE LESIONS

These lesions are a direct result of a chronic irritation physical, chemical or irradiation.

Linea Alba Buccalis

Most mouths have localized whitish papular area due to sharp cusp of some of the teeth on the buccal mucosa, which we term as frictional keratosis. When patient is habituated in biting his own cheek frequently then we see a linear extension of this frictional keratosis at the Occlusal plane which we call as the Linea alba buccalis traditionally and is a self reversing lesion once the patient stops his self cheek biting habit.

Leukoedema

In Indian population the people who indulge in smoking bidis and cigarettes for over five years continuously tend to show diffuse white areas of mild corrugation, this whiteness vanishes once the cheek is stretched. Many authors believe that the relationship between the tobacco smoking and Leukoedema is not clear. This lesion does not require any treatment as such, the counseling to reduce and stop the tobacco use is always on the main agenda of a good dental practitioner.

Cheek Bite Ulcer

Self-induced—In periods of anxiety like examinations, preparing for a sports meet etc. some individuals tend to indulge in chewing their cheeks to the extent of causing ulcer that is traumatic in nature. Personal history of the patient and his transient period of stress may be noted and local pain relief may be prescribed by Ointment Mucopain®, Chlohex ® gel (cholorhexidine gel) to apply locally so that healing is promoted. Any chronic ulcer that shows no sign of healing in three weeks time should be biopsied and referred by specialist advice.

Traumatic Ulcer

Clasps of the partial dentures delivered by denturists, some orthodontic appliances, sharp teeth and overhanging

fillings all may physically irritate the oral mucosa and cause ulceration. The pain and super-infection with local commensals usually brings the patient to the dental practitioner.

Eosinophilic Ulcer

Vélez A et al (1997)[1] have introduced a diagnostic label termed as EUOM or Eosinophilic ulcer of the oral mucosa. They described one such lesion on the tongue which was benign and self-limiting. Microscopically it contains structure of non-specific ulcer with a very rich infiltrate of eosinophils. Trauma has been attributed as a primary cause for this EUOM.

Ficarra G et al (1997)[2] mention about Eosinophilic ulcer on the tongue of a 72 year old male and mention injury as the primary cause of this ulcer. They explore the possibility that this type of ulceration may be associated with immunostain with CD30 antigen.

Riga-Fede Disease

Uzami SM et al (1999)[3] mention about neonate suffering from sublingual ulceration due to pre-eruption of the deciduous teeth causing feeding problems and also ulceration of the mother teat**.** This has been termed as the Riga Fede disease. This traumatic ulcer is self-healing once the offending natal teeth are extracted.

Betel Chewers Mucosa

Reichart PA and Phillipsen HP (1998)[4] have described the prevalence of BCM or Betel chewer's mucosa as characterized by brownish –red discoloration of the oral mucosa with tendency to desquamate. Women have been said to be more affected and prevalence figures varying from less than 1 to 60 percent in different countries of south East Asia have been described. This lesion is reactive to the chemical constituents of the traditional "Pan Chewers". This type of a mucosa may be seen by dentists across the country where chewing in practiced regularly and culturally.

Chemical Ulcers

Ulcers are caused by chemical contact with aspirin tablets and a case of large ulcer resulting from ferrous sulphate tablet contact is mentioned by Fern´andez-Viadero C et al (1998).[5]

Reactive Fibrous Hyperplasia

Whenever there is a physical irritation and it is of low grade and the oral mucosa is young and exuberant, instead of breaking into an ulcer, the oral mucosa get stimulated and a reactive fibrous hyperplasia results. These pedunculated tough lesions have been also called as reactive fibroma by some clinicians. Two extensive surveys of biopsies one from Brazil of 1018 biopsies (Maia DM et al[6] 2000) and from Singapore of 2057 biopsies (Tay AB[7] 1999), reported that inflammatory fibrous hyperplasia was commonest of the lesions in these series.

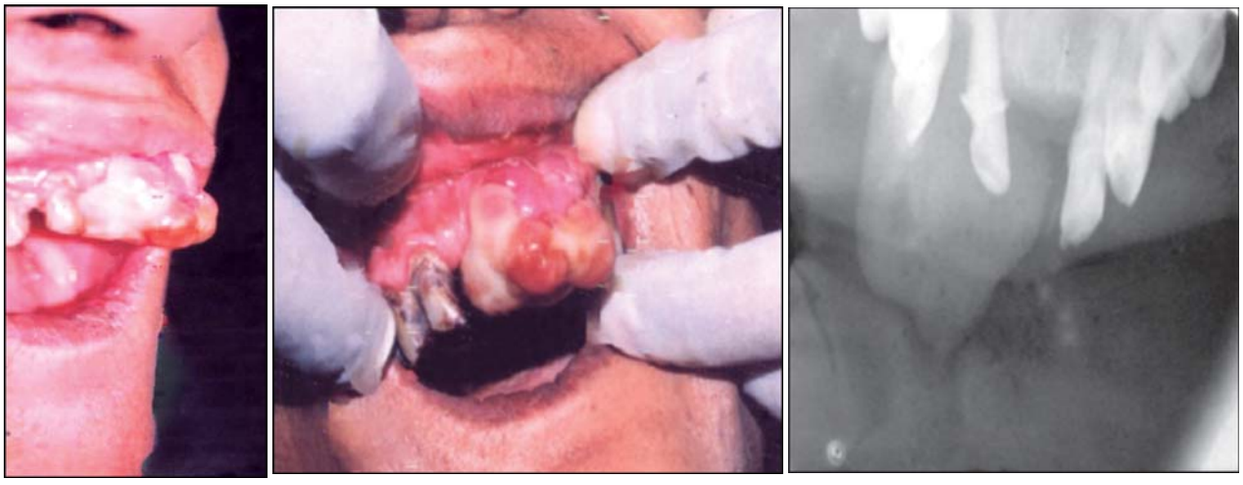
IPEH

de Courten A et al (1999)[8] have described IPEH (intravascular papillary endothelial hyperplasia) as a vascular reactive lesion with specific histopathological features and grouped them in vascular malformations group. Male predominance was observed and most lesions were seen on inner mucosa of the lip.

IPHP (Inflammatory papillary hyperplasia of the palate), Palatal papulosis, Papillary hyperplasia of the palate; Papillary overgrowth usually seen under a full denture maintained poorly or when medically compromised patient gets overgrowth of the candidal infection. It has also been called 'leaf fibroma' due to the fibrous hyperplasia getting flattened under the upper denture. Simple excision, non-wearing of dentures for two weeks and anti-fungal treatment usually gives uniformly successful results. Patient should be also given proper hygiene instructions in maintenance of the denture.

Brown AR et al (1997)[9] has commented that viral laboratory techniques indicate that human papilloma virus HPV is associated with papillary lesions of the palate. They also mention of a particularly huge lesion which they treated using free soft tissue grafts with very good results.

This p53 and PCNA (proliferative cell nuclear antigen) are cell-cycle regulators. When they are over-expressed they are considered as indicators of malignant potential. Studies by Kaplan I et al[10] from Tel Aviv University have



FIGURES 20.1A to C: Shows a large pyogenic granuloma, arising in the upper labial gingiva,which had grown on the teeth and covered it completely. The radiographic shows the soft tissue shadow of the tumor and heavy bone loss in relation to the teeth. (Nillofer S, Prasanna K, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

found three times the normal cells stained for p53 and twice the normal cells stained for PCNA in 12 lesions of IPHP. They have cautioned against hasty conclusions being drawn regarding the pre-malignant potential of IPHP. Denture trauma and candidal infection continue to be believed as primary causative factors. Considering the fact that many in India are involved in chutta (reverse smoking) and clay pipe smoking any lesions of the mouth, which show suspicious activity, must be excisionally biopsied.

Pyogenic Granuloma (PG)

This appears commonly on the gingiva as cherry red to purple spherical swelling which is painless and bleeds to touch. The term PG is a misnomer since this is a reactive lesion and normally does not show any pus formation. Histologically it shows a rich proliferation of capillaries and may be called as the Reactive capillary lesion. When it occurs in pregnancy it is termed as pregnancy granuloma. It develops due to poor local oral hygiene and changes in the esterogen levels in such cases. Removal of the lesion and maintaining of meticulous oral hygiene will prevent its recurrence. Raulin C et al[11] 1997 treated 13 consecutive patients with CO2 laser and found that this is a fast and bloodless way to treat the PG. Pyogenic granulomas have also been reported in nasal cavity and on the Penis (Tomasini C et al[12] 1998) (Fig. 20.1).

Giant Cell Reactive Lesion

In young patients usually commonly the peripheral blue red soft tissue lesions seen on places of local irritation when biopsied turn out to have a rich proliferation of giant cells in the stroma. Clinically many of them look like pyogenic granuloma or even a reactive fibrous hyperplasia, but it's the Histopathological diagnosis that clinches the label to be used (Fig. 20.2). Tiffee JC and Aufdemorte TB[13] have done classical study to trace the lineage of these giant cells in the oral lesions and opine that they may arise from precursor cells related to the granulocyte/macrophage line, and may originate from mononuclear cells that express markers for both macrophages and osteoclasts.

Two main varieties of GCRL have been noted. Those that occur peripherally and those that occur within the bones. They are termed as peripheral and central giant cell lesions. Many workers believe that these are related to the alteration in the functioning of Parathyroid and thyroid metabolism that causes excessive removal of the calcium from the bones resulting in such lesions. If a central lesion is noted in any patient then Alkaline Phophatase and Serum Calcium studies in addition to routine hematology will give an indicator whether systemic factors are involved.

Routine excision or Laser treatment gives good relief and when indicated endocrinologist needs to be consulted on whether Calcitonin therapy is necessary.

Many times the adjacent teeth like the deciduous or permanent may have to be sacrificed as mentioned in a case by Bhat SS et al[14] from Mangalore. A large peripheral giant cell lesion was interfering with occlusion and when the enucleation was taken up the involvement of the deciduous and the permanent premolar was necessary. They mentioned that poor oral hygiene and Xerostomia were risk factors for this lesion. There was no recurrence.

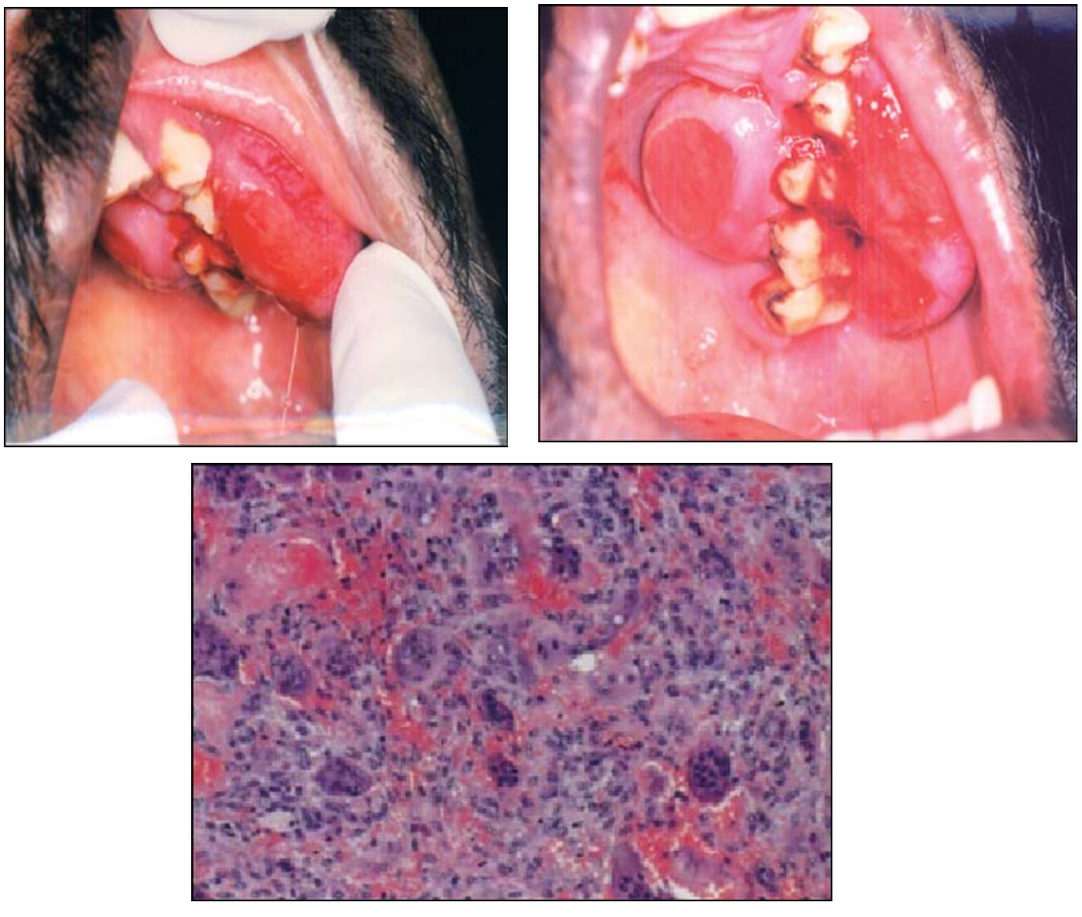
There is a group of investigators who believe that esterogenic activity affects the growth of the peripheral giant cell lesions in oral cavity. Gunhan M et al[15] have used immunostaining techniques and studied 26 peripheral giant cell lesions, estrogen receptor positivity was found in statistically significant number of them.

Some of these lesions show exuberant and uncontrolled growth and show histological changes similar to malignant cells, the term malignant giant cell lesions has also been noted. Immediate and surgical intervention with wide excision is recommended as treatment of choice.

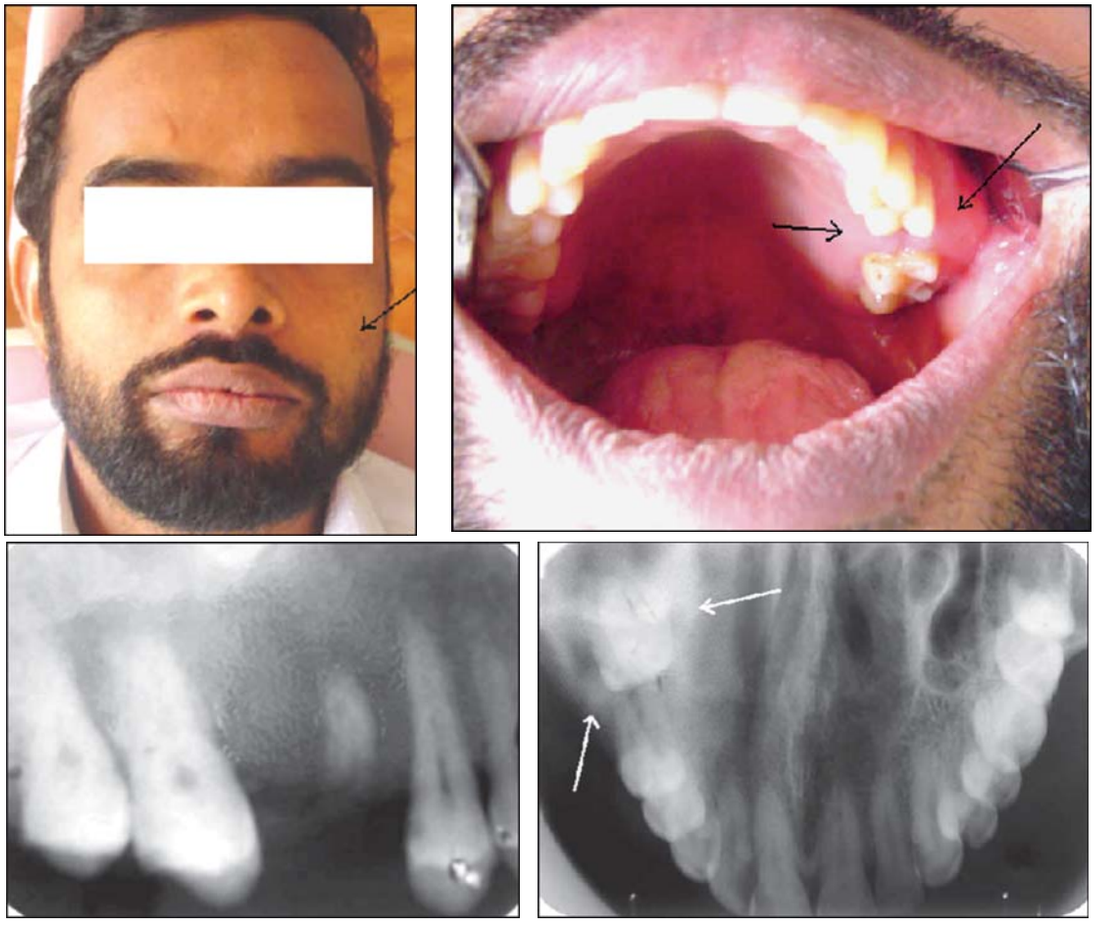
Bodner L et al (1997)[16] studied 79 patients of peripheral giant cell lesion and found that larger lesions were more likely in women, with poor oral hygiene and relatively more dryness of mouth.

Fibrous Dysplasia

Fibrous dysplasia is a hamartomatous condition characterised by replacement of normal bone with immature fibrous tissue. It is a sporadic condition associated with



FIGURES 20.2A to C: A 37-year-old male who had high risk sexual behavior history and HIV positive reported with painless swelling in the maxillary premolar molar region. The rapidly growing lesion was just 7 months old. Histopathology revealed several giant cells in a stroma of proliferating blood vessels and chronic inflammatory cells. (Leela KP, Rohit M, Bailoor DN 2002, Yenepoya Dental College Hospital, Mangalore)



FIGURES 20.3A to D: Showing 38-year-old medically fit male patient with complaint of asymptomatic swelling in the left upper posterior arch. Intraorally bicortical expansile lesion. IOPA view shows classic 'Ground Glass' appearance and occlusal view shows localized bicortical expansion (Beena K, Omal PM, Bailoor DN 2004, Yenepoya Dental College Hospital, Mangalore)

mutation in GNAS 1 gene (Guanine nucleotide-binding protein, alpha stimulating activity polypeptide 1). The clinical presentation can be localized form referred as 'Monostotic' or diffused with involvement of multiple bone referred to as 'Polyostotic'. Syndromic forms are associated with cutaneous pigmentation and endocrine disturbance and multiple bone involvement. Monostotic is more common (70%) (Fig. 20.4).

Clinical features: Condition is diagnosed in second decade, with equal sex predilection. Usually seen as a painless swelling with more maxillary involvement (Figs 20.3A and B).

Chief radiographic feature is a ground glass opacification produced due to poorly calcified bony trabeculae arranged in disorganized pattern. Margin blends with surrounding normal bone. The early lesion may be radiolucent. Mandibular involvement causes superior displacement of inferior alveolar canal (Figs 20.3C and D).

Histopathologically we see a Chinese letter appearance.

Large lesions are usually surgically resected or recontoured. Radiation therapy is contraindicated due to probability of sarcomatous change.

Radiation Mucositis

Dental surgeon working for large hospitals with full-fledged oncology units will be called upon to consult regarding the post-radiation complications. Radiation

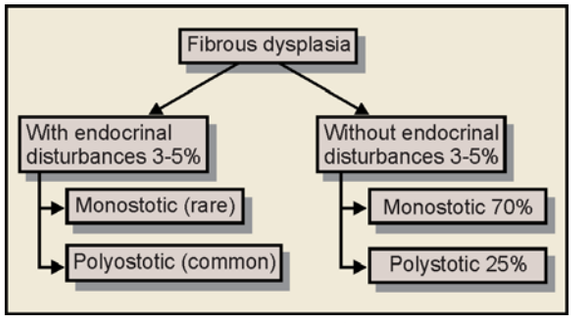


FIGURE 20.4: Classification of fibrous dysplasia

mucositis is one of them. Use of Mucopain® local anesthetic creams and Use of artificial saliva is to be routinely recommended. Nutritional supplementation with sufficient zinc and other micronutrients is indicated. If there are teeth in the oral cavity then there additional protection using 4 percent sodium fluoride varnish and use of fluoride mouth wash (S-flo®) four to six times a day will definitely help.

- *Ad Film (Hydroxy-Propyl-Cellulose based Film)*: Oguchi M et al (1998)[17] have devised a water soluble polymer film called as the AD film which contains anesthetics and antibiotics for treatment of the radiation mucositis in the mouth. The base they used is hydroxy-propyl-cellulose. After using this film on 25 patients they opine that this film helps in reducing secondary infection, improves patient comfort and allows peroral feeding.
- *Antibiotic Lozenges:* Fifty four patients receiving radiation therapy were put on a random controlled trial to test efficacy of non-absorbable antibiotic lozenge by Okuno SH et al[18] 1997. Mucositis was less in the group, which had the antibiotic lozenge, and further studies have been recommended using different antibiotics in more patients.
- *Sucralfate Oral suspension* given as six doses of one gm gave a significant relief to the post irradiation Mucositis. Etiz D et al[19] 2000 used it on forty-four patients with random control trial. They reported no significant side effects.
- *Mouth washes* containing granulocyte macrophage-colony-stimulating factor (GM-CSF). Twelve patients using the GM-CSF containing mouth wash showed significantly quicker healing and lesser pain and discomfort in the study by Rovirosa A et al [20] 1998 but they mention the need for Random controlled trials before the doses and frequency can be standardized. A total of 300 micro gms of GM-CSF in 250 cc of water for 1 h of mouth washing was prescribed by them.

In case of severe pain inj pethidine or morphine for pain and use of pilocarpine for stimulation of the salivary flow is indicated.

Torus Mandibularis—Maxillaris

These two terms are used for exostoses of bone in mandible and maxilla. No obvious cause is established in most cases. Torus palatinus usually appears as single, double or tetroid bony nodule in the middle of the palate. In most patients these stop growing by puberty. In the mandible these out growths are bilaterally symmetrical and grow in the mental-premolar region on the lingual side. These lesions have no health hazard and when dentures are made proper relief needs to be given.

HAMARTOMAS

Hemangioma- Lymphangioma- Pigmented cellular nevus

Hemangioma

These are vascular malformations, which occur at birth. The birthmarks also are variants of this lesion. They are more common in females in ratio 2:1. More than half the cases are noted clinically before the first decade of life. They appear on skin as macular areas red to pink to blue and sometimes as elevated lesions with bruit. They appear to grow in size up to puberty and then they stop growing. Conversion into true neoplasm's is rare (Fig. 20.5).

Most hemagiomas need to be removed for the esthetic reasons. They are best treated by either cryosurgery or laser surgery. No biopsy must be done in private dental clinic for such lesions.

When meningeal involvement is noted, it is associated with epilepsy and mental retardation known as the *Sturge-Weber Syndrome*.

Histologically: They are termed as either cavernous, capillary or mixed. In fact what is histologically reported as the capillary hemangioma of the peripheral oral tissues are many at times actually reported clinically as 'pyogenic granuloma'.

FIGURE 20.5: Shows healing capillary hemangioma of the lip in a nine-year-old female child who was attempted for treatment at a local dentists place during which there was uncontrolled bleeding. (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

Another entity termed as the central hemangioma is seen rarely in the jaws. It is recognized purely by its radiographic appearance. Clinically it appears as a painless bony hard swelling, which may cause loosening of the teeth. Radiographically it appears as an irregular osteolytic lesion, which is multilocular. The trabeculae are fine in most cases and honey combed appearance has also been described.

Phleboliths are fine calcifications seen in the radiolucencies of the hemangiomas.

Contrast arteriography helps to outline many of the hemangiomas prior to any surgical decisions being taken. Many clinicians prefer to use Sclerosing solutions prior to any attempt at cryosurgery. Wide surgical excision using cryosurgery seems to be the treatment of choice.

Lymphangioma

Lymphangiomas are congenital lesions consisting of mass of Lymphatic vessels and spaces. Most are detected at birth and when they are large they may need immediate intervention. But sometimes-small nodular Lymphangiomas may be seen up to puberty. Some investigators believe that Tongue may be affected and they may cause macroglossia. Color of lesions varies from blue to pink depending on its depth in the oral tissues.

Macrocheilia may be caused by lymphangioma of the lips.

Lymphangioma of the neck is known as the Cystic Hygroma and these can cause respiratory distress and need emergency care.

Histologically: Characterized by endothelial lined lymphatic spaces of various sizes. No major conversion into malignant varieties is reported and most of times the lymphangioma needs to be surgically excised for cosmetic reasons, rarely for alleviating respiratory distress.

Differential diagnosis: Small lesions need to be differentiated from mucoceles and mucocutaenous lesions.

Nevus

Nevus usually means congenital lesion or an hamartoma made up of melanocytes and their derivatives. Terms like neuvo-cellular nevus have also been used. The origin of nevus cells is not known but most authors postulate that there is some relationship between the melanocytes and the nests of nevus cells and others mention neural crest origin.

Intraorally common site is palate and it occurs as a pigmented papular lesion; it can occur almost anywhere intraorally.

Regezi (1989)[21] mentions about four types of nevi histologically—

A. *Junctional nevus*
B. *Compound nevus*
C. *Intradermal nevus*
D. *Blue nevus*

Depending on the location of the nevus cells the above categories are recognized.

Junctional is when the cells are predominantly at the basal layer of the epithelium.

In the submucosal area the term intradermal has been used. When both the above groups are seen in the same specimen then we term it as compound nevus.

When cells are seen very deep in the connective tissue and are spindle shaped then the term blue nevus is used.

In oral cavity intra-mucosal nevi are the common variety and blue nevi are also seen.

(* Please see figure in Chapter 14).

For the practicing dentist what is important is that if he sees any pigmented papular lesion which has been there for a long time without any change, then leave it alone, but keep the patient on regular recall (every two months at least). If there are signs that the lesion is growing in size or ulcerating then referral to a Oral Physician and Radiologist is indicated. Differential diagnosis must include Amalgam tattoo, Varices in the under part of the tongue, OMM Oral Melanotic Macule, and Melanoma.

BENIGN TUMORS

Epithelial Origin—Papilloma—Keratoacanthoma

Papilloma

Papilloma is actually a group of lesions, which range from a true reactive papillomatous hyperplasia to truly benign tumor the 'papilloma' (Fig. 20.6).

In most of the lesions two risk factors have been identified, local irritation and HPV subtype 11 virus infection. Complete excision and removal of the local factors is usually met with success. Always remember to send the tissue for Histopathological evaluation.

FIGURE 20.6: Showing the papilloma on the palate (Kartikeya Patil, Mahima Patil 2004 JSS Dental College, Mysore)

Keratoacanthoma

This is a lesion, which lies on the borderline between the reactive and benign and is mainly postulated to be caused on sun-exposed part of the skin. Major risk factors appear to be sunlight, viruses and trauma or local irritation. Many lesions are known to occur at the vermilion junction of the lips. They may appear singly or multiple. It begins as small red macule, which grows into a firm nodule with tip of the nodule containing a plug of keratin. In four to six weeks time this grows radially and the central portion gets a little depressed with a central keratin core. It gives it a small moon crater like appearance. There is no induration and unless the patient scratches himself, and infects it, there is no lymphadenopathy.

Lou YR et al[22] 1999 mention there is a protective effect of black tea drinking in development of various papillomatous and keratoacanthomatous lesions in experimental mice. These mice were irradiated to a dose of Ultraviolet B light and then given green and black tea in the experimental group. Green tea and black tea inhibits the formation of papillomatous and keratoacanthomatous lesions in animal studies (SKH-1 type mice). The inhibitory effect was not noted in decaffeinated teas.

Histologically there is great similarity between the well differentiated squamous cell carcinoma and the keratoacanthoma. The differentiating points are smooth and balanced infiltration seen in the crater like central portion of the lesion. The epithelium shows a raised crater like lipping and the oval dip contains a central keratin plug seen clearly in low magnification. A more detailed histological debate is beyond the scope of this basic diagnostic pointers chapter.

Rare intraoral lesions are associated with Fordyces granules like lesions containing ectopic sebaceous glands.

Many lesions undergo spontaneous regression leaving a scar. But as a rule it is good idea to follow up the lesion and if slightest doubt regarding its edges being indurated occurs early total Excisional biopsy is the treatment of choice.

Ishida CE and Ramos-e-Silva M[23] 1998 used a cryosurgery liquid nitrogen spray or cryoprobe to treat several types of lesions including keratoacanthomas and actinic cheilitis and found it to be a inexpensive and safe outpatient technique for treatment.

- Connective tissue origin—Fibroma—Lipoma
- Chondroma—Chondroma variants (Fig. 20.9)
- Myxoma—Mixed variants like Benign chondroblastoma—Chondromyxoid fibroma, etc.

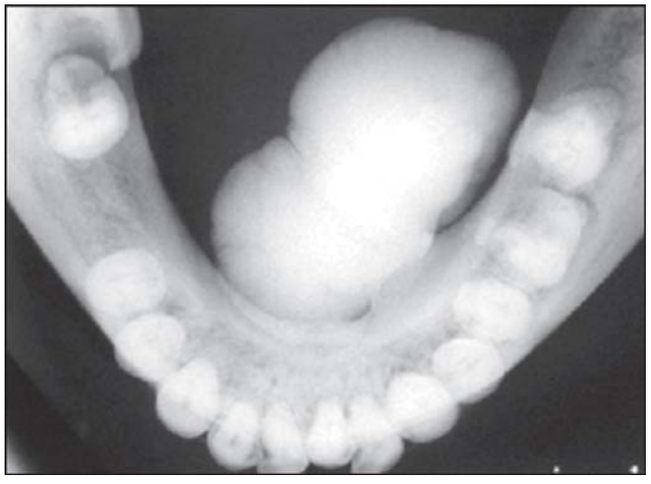
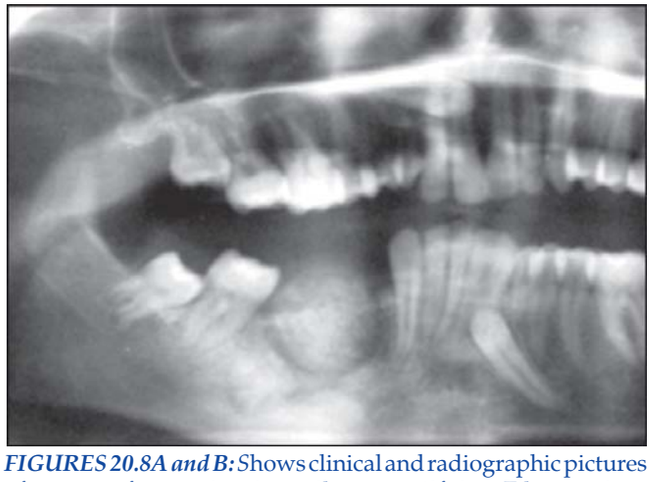


FIGURE 20.7: Showing occlusal radiograph of mandible with the radiopaque area extending lingually diagnosed as osteoma (Patil Mahima K 2004, JSS Dental College, Mysore)



FIGURES 20.8A and B: Shows clinical and radiographic pictures of a case of recurring juvenile cementifying Fibroma in a seventeen year old male patient who had severe pain and swelling in that region since one month (Nillofer S, Prasanna K, Bailoor DN 2003, Yenepoya Dental College Hospital, Mangalore)

Fibroma-Lipoma

Any slow growing nodular lesion in the oral cavity which has a tough fibrous feel and continues to grow even after removal of the local irritation or trauma would raise suspicions of a fibroma. Actually the difference in consistency and Histopathological picture gives us a firm footing to make a diagnosis. Lipoma has oily, springy feel, which is characteristic. The giant cell lesion is of course cherry red or deep blue bleeds easily to touch and only the biopsy gives us the giant cell filled lesions in fibrous or capillary stroma.

With the refinements in staining techniques and immuno-histochemical methods newer labeling system for diagnosis are emerging.

Rousseau A et al (1999)[24] present a nodule in the Buccal mucosa of a 60-year-old male diagnosed as Giant cell angio-fibroma. They mention that diagnosis is essentially histologic and immunohistochemical.

Chondroma—Osteoma Variants—Mixed Variants like Benign Chondroblastoma—Chondromyxoid Fibroma, etc.

In this group of lesions if the clinical feel is essentially tough and cartilaginous, think of chondroma, hard bony feel think of all the osteoma variants and so on. Clinically all these lesions can appear variously as slow growing

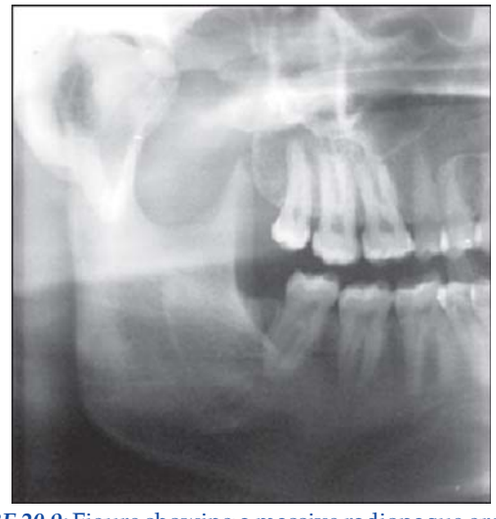


FIGURE 20.9: Figure showing a massive radiopaque growth in the condyle of the right side which was reported histopathologically as Chondroma variant (Mahima Patil, Kartikeya Patil JSS Mysore 2004)

nodules in various parts of the oral cavity. The last word is that of the histopathologist combined with the radiographic features. The private practitioners who are interested in pursuing these lesions in more details are requested to peruse the excellent texts on oral pathology currently available.

MALIGNANT TUMORS

Epithelial Origin

- Basal cell carcinoma
- Squamous cell carcinoma
- Adenoid squamous cell carcinoma
- Transitional cell carcinoma
- Verrucous carcinoma

Above five lesions represent the neoplastic changes in the oral mucosa component and has been described in details in the Chapter 17 on Oral Cancer.

Look for systemic indicators of emaciation, loss of weight more than 10 percent within 3 months, and ulcerative growths, which are rapidly growing, and widespread cervical lymphadenopathy, which becomes hard and fixed to underlying tissues. A quick biopsy and referral of all suspicious lesions should go a long way in giving satisfaction to primary care dentist. All of the above lesions have typical histological and immunohisto-chemical points of recognition.

Malignant Melanoma

Luckily only 1 percent of the melanomas occur in the oral cavity. The palate is the most common site for any pigmented nevus like lesion, which is growing in size and ulcerating must be suspected as malignant melanoma. Clinically the color of lesion can vary between brown, black and blue. Very irregular borders and rapid spread are the cautionary findings. Superficial spreading, nodular and Acral-lentiginous are the three types seen intra-orally. Each has typical Histopathology. Since many of the melanomas appear fairly innocuous early biopsy of all the pigmented lesions should be a rule.

Tanaka N et al (2004)[30] have discussed recently about the Amelanotic oral malignant melanoma (AOMM) which is a rare tumor that is difficult to diagnose. They have mentioned that immunohistochemical reactions, and electron microscopy were reasonable aids to diagnosis in such cases.

Garzino-Demo P[31] et al 2004 analyzed ten new cases of oral melanomas and found that average age of the patients in their series was 64.3 years and 60 percent of the patients were males. Six of the ten lesions were in soft hard palate and rapidly growing . Radiation and adjuvant therapies have been utilized with varying successes in this series.

Connective Tissue Origin

Osteosarcoma-Chondrosarcoma

Osteosarcomas of the jaws constitute approximately 4 to 6 percent of the total sarcomas according to different series. They peak in incidence mainly in the first two decades there is a second peak of increased incidence around fifth decade. We could look at osteosarcomas as those occurring in the bone itself, and rare ones occurring in the soft tissues.

First group (Figs 20.7 and 20.8)

a. Periosteal surface is the origin of the tumor
b. Arising in the medullary cavity of the bone.

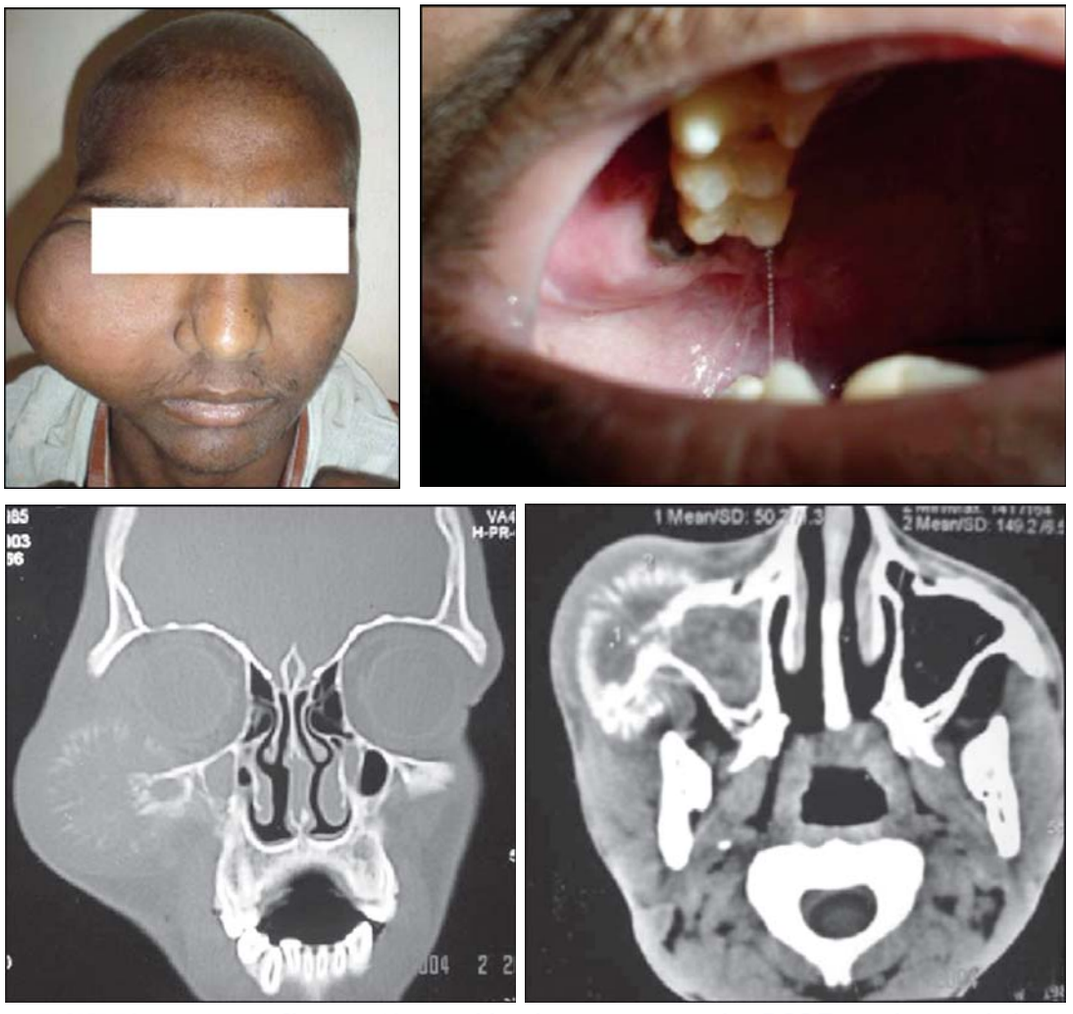
Risk factors: There are a few conditions, which increases the risk of osteogenic sarcoma development they are:

- Pagets disease
- Osteogenesis imperfecta
- Fibrous dysplasia
- Chronic osteomyelitis
- Multiple osteochondromas
- Previous irradiation for oral cancer treatment etc.

Equal predilection for mandible and maxilla. Sixty percent of mandibular lesions seen in the body. Fast growing, nodular, bony hard with paresthesia of the lip, side of the face or other facial areas depending on the tumor location. The chief complaint of the patient is usually less than three to six months duration (Figs 20.10A and B).

Piattelli A and Favia GF[26] 2000 mention that osteogenic sarcoma of the jaws are very different from the those of the long bones. They tend to occur at an older mean age, pain and swelling are more typical, and prognosis is more favorable using multimodality treatment.

Radiographically most osteosarcomas show irregular radiolucent areas, widening of periodontal ligament



FIGURES 20.10A to D: Showing 18-year-old male patient reported to RCC Trivandrum with the complaint of swelling on the right side of the face, which was diagnosed as osteogenic sarcoma of the right maxilla (Courtesy: Ramdas K, Dept of Radiotherapy, RCC Trivandrum 2004)

locally, 'tooth hanging in air appearance', large areas of destruction have been described as moth eaten appearance, these findings can easily correlated with tingling sensation and paresthesia when tumor invades the neurovascular bundles supplying the facial region (Figs 20.10C and D).

Juxtaperiosteal tumors give rise to periosteal reaction which has been characteristically described as the sun-ray appearance due to radiating radiopaque lines in the radiolucent regions.

Histologically malignant osteoid production by spindle cell stroma is one of the appearances. Many senior pathologists describe the osteogenic histology as one, which is either dominated by osteoblastic elements, chondroblastic elements or fibroblastic elements in the spindle cell stroma of sarcomatous background. The detailed Histopathology of this lesion is full of debate and beyond the scope of this diagnostic text which is more practitioner oriented.

Pandey M et al (2000)[25] in a retrospective study from Trivandrum Regional cancer center which recorded ten cases of soft tissue sarcomas in the time period between 1990 and 1998. Mean age given was 31 years, 70 percent of the patients were male, Rhabdomyosarcoma, and Spindle cell sarcoma were the most common in the

series and they mentioned that multimodality treatment gave the best results. Most clinician prefer to start with radical surgery followed by chemotherapy in case of recurrence.

Chondrosarcoma of the Jaws

Jaw chondrosarcomas are rare, aggressive, and show local recurrence. Around 10 percent of the tumors metastasize to lungs or long bones. Hard tissue chondrosarcomas and Mesenchymal chondrosarcomas are some of the variants reported in the pathology literature.

Anterior maxilla is the site in 60 percent of the some of the series studied. Swelling, pain, loosening of teeth and paresthesia are some of the findings in facial region.

Radiographically a poorly circumscribed radiolucency which is usually multilocular with dense calcifications present. But radiography alone without Histopathology cannot confirm the diagnosis (Fig. 20.9).

Histopathologically—The chondrocytes are pleomorphic, binucleated and show heightened mitotic activity. They are in Myxoid stroma and show wide variation in degrees of calcifications.

Vencio EF (1998)[27] in their classical study of nineteen cases of mesenchymal chondrosarcomas found that average age in this series was 19 years with 84 percent patients below 30 years of age.

Histologically, the classic picture of a bimorphic tumor, composed of islands of well differentiated hyaline cartilage juxtaposed to a small cell undifferentiated malignancy.

Resection, including hemimandibulectomy and hemimaxillectomy, was the main treatment chosen by the clinicians in this study.

Lockhart R et al (1998)[28] reported four cases of Mesenchymal chondrosarcoma (MCS) and attributing to their rarity, mentioned that after being first described by, Lichtenstein and Bernstein in 1959 only a total of 46 cases were published globally.

Gorsky M and Epstein JB (2000)[29] analyzed 34 hard tissue sarcomas of the jaws over a 29 year period and found 11 chondrosarcomas and 23 osteosarcomas. Mean age seen at diagnosis was 40 years in this series. Combination of the Radical surgery with either chemotherapy or radiation continues to be analyzed by the evidence-based researchers.

Lymphoreticular System

Lymphoma-Multiple Myeloma

Lymphomas are a diverse group of malignant lesions, which are characterized by monoclonal proliferation of the lymphoid cells.

One of the ways to classify it is—

- *Cutaenous lymphomas*
- *Systemic lymphomas*

Hodgkin's Disease—Non-Hodgkin's Lymphoma—Burkitt's Lymphoma

If a patient presents with generalized debility, weight loss more than 10 percent, varied skin lesions and nail changes together with cervical, inguinal and axillary lymphadenopathy which is rubbery and non-tender. Suspect Lymphoma group of disorders.

Demonstration of cells positive for monoclonal antibody Ki-1 is supposed to be diagnostic. Ulcers are present usually in the tonsillar lymphoid tissue area and posterior palate. (Gingiva is rarely involved—helps to differentiate clinically from the leukemia and agranulocytosis).

The Burkitt's lymphoma is usually seen as rapidly growing enlargement of mandible in children below 3 years of age on the African continent. It is a typical B-cell lymphoma which ulcerates in the mouth and causes loosening of the teeth. Emaciation and death is occasionally seen. Insect carrier which transmits Epstien Barr virus has been implicated.

Microscopic picture of sheets of dark staining round cells with many clear spaces with one or more pale cells gives rise what is called as the 'starry sky appearance'. Chemotherapy is usually the treatment of choice.

Multiple Myeloma (MM)

It is a malignancy of the plasma cells in which they produce only one type of immunoglobulin in exaggerated quantities so much so that the cells are crowded out of the circulation. RBCs getting crowded out may present as severe anemia, and thrombocytopenia can present as bleeding diathesis as the first symptom. Most patients complain of bone pain and tenderness (Figs 20.11 and 20.12).

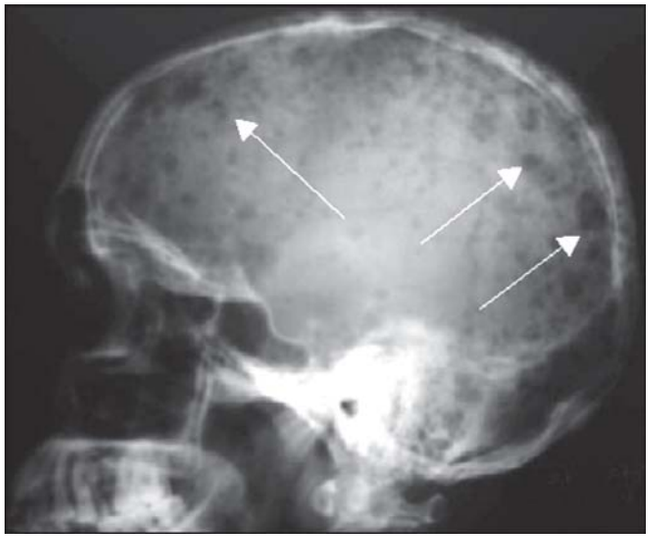


FIGURE 20.11: Showing characteristic punched out appearances of the skull in case of Multiple Myeloma. (Courtesy: Ramdas K, Dept of Radiotherapy, RCC Trivandrum 2004)

FIGURE 20.12: Showing 63-year-old female with splitting headache of 2 years duration. Skull radiographs gave the first hint to the abnormality and the proteins present in the urine confirmed the diagnosis of Multiple Myeloma (Courtesy of Mody RN GDC, Nagpur 2004)

Diagnosis is based on

a. Radiography—In which characteristic punched out appearances of the skull and jaws is almost diagnostic
b. Increased ESR
c. Abnormally high immunoglobulins
d. Bence Jones proteins in the Urine
e. Bone marrow biopsy
f. Serum electrophoresis

This is a serious disorder which will be treated only by hospital based dentists. The five year survival rate is less than one in five. Dental treatment is complicated by anemia, bleeding diathesis and postoperative infections.

Multimodal chemotherapy is supposed to be the preferred treatment. But research continues in better treatment options around the world.

Amaral L et al (2003)[32] in a retrospective analysis of 81 cases found that MRI identifies bone marrow abnormalities and invasion of adjacent tissues at an early stage. Therefore, it is an essential method when it comes to properly evaluating skull lesions.

Thus, now the stress in on more sensitive imaging modalities like CT scan and the MRI for in-depth evaluation of the multiple myeloma lesions.

MUSCLE AND NERVE TISSUE TUMORS—THEY ARE RARE AND MAY BE STUDIED FROM SPECIALIZED TEXTS

CONCLUSION

The diverse group of lesions discussed in this chapter range from the reactive, to hamartomatous to benign to the fatally malignant. This chapter is merely indicative of how extensive research and literature is being developed on each lesion. The primary care dentist must just know how to distinguish between the reactive and the malignant. Remember when in doubt, histopathology, radiography and hematology will come to your help. Referral to Oral Physicians is the next logical choice.

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Bailoor DN, Gopakumar R

21 Halitosis

INTRODUCTION

The word halitosis derives from Latin, 'halitus=breath' and 'oris disease = mouth condition' literally meaning bad breath. Halitosis is a symptom, not a disease. It is one of the commonest clinical problems encountered in dental practice. Often the cause is evident in the oral cavity, like an open carious lesion, periodontal abscess or pericoronal abscess. But if intra-oral causes are not present, systemic contributing factors should be suspected; like lung abscess, acid peptic disease, suppurative pharyngitis etc. In some patients who apparently have good oral hygiene there is a feeling that their mouth stinks and they persistently go from doctor to doctor who tells them that there is nothing wrong and that they don't have any halitosis. In such cases delusional halitosis should be suspected.

DEFINITION

Halitosis refers to bad breath, emanating from the mouth. It is also called as fetor exore or fetor oris.

CLASSIFICATION AND CAUSES

There are various causes of halitosis. It can be broadly classified into:

- Physiology
- Pathologic
  - Oral
  - Extraoral or systemic
- Psychogenic

Physiologic Causes

Pregnancy, Starvation: 'Hunger breath' is due to putrefaction of pancreatic juices in the stomach. In Xerostomia, halitosis may be a clinical feature as there is a lack of cleansing action of saliva.

a. Food:
   i. Spicy food
   ii. Degraded waste products of ingested protein substances may be eliminated through lungs. Common examples are meat containing fat and volatile fatty acids. Onion, garlic, alcohol and fish.
   iii. Vitamin 'C' deficiency.

b. Drugs: Some of the drugs causing halitosis are:
   i. Isosorbide Dinitrate
   ii. Drugs containing Iodine and Chloral Hydrate
   iii. Diuretics
   iv. Antineoplastic drugs such as Methotrexate, Adriamycin Bleomycin may cause mouth ulcers, candidiasis, periodontal breakdown and dry mouth leading to halitosis.

Pathologic

a. Oral—Tongue coating, poor oral hygiene, gingivitis, periodontal disease, pericoronitis, food impaction, extraction wounds, dry socket, anerobic infection, endodontically treated patients during treatment may feel the smell of eugenol or other medicaments. Rule out oral malignancy, tonsillitis, xerostomia
b. Extraoral or systemic causes
   i. Blood and blood disorders: Patients with bleeding disorders such as hemophilia, thrombocytopenia, there may be decomposition of blood resulting from spontaneous bleeding leading to bad breath. Patients with anemia also have halitosis.
   ii. Ear: Otitis media
   iii. Respiratory system:
      1. Nose—Rhinitis
      2. Sinus—Chronic sinusitis
      3. Larynx—Laryngitis
      4. Pulmonary—Lung abscess, bronchitis, bronchiectasis, TB

      Patients with lung abscess or bronchiectasis have breath that is described as that of odorous rotting meat.
   iv. Gastro-intestinal tract:
      1. Pharynx—Pharyngitis
      2. Esophagus—Reflux esophagitis
      3. Stomach—Peptic and duodenal ulcers, hemorrhage, indigestion
      4. Liver—Severe hepatic failure leads to 'fetor hepaticus'—characterized by fresh cadaver smell.
   v. Cardiac:
      1. Rheumatic fever imparts an acid sweet breath.
      2. Cor pulmonale.
   vi. Renal: Renal failure, nephrotic syndrome, uremia kidney failure and uremia result in smell of ammonia or urine.
   vii. Endocrine and metabolic:
      1. Hypothyroidism and hyperthyroidism
      2. Patients suffering from diabetes mellitus may have acetone breath due to excretion of acetone through lungs.
      3. Children and adults have sweet pleasing breath. Relatively low basal metabolic rate during sleep results in decreased salivation.

The decreased saliva flow during sleep leads to accumulation and putrefaction of epithelial cells, food debris and saliva resulting in an unpleasant morning breath.

Psychogenic

Delusional halitosis is a type of psychosis where the patient thinks that he has halitosis and seeks professional help. Such patients may repeatedly visit their dentists and demand oral prophylaxis. These patients may show meticulous care in maintaining their oral hygiene. Counseling, anxiolytics and antidepressants are known to help. Referral to an Oral Medicine Specialist is indicated.

HALITOSIS PRODUCTION

Halitosis production has been attributed to the production of sulfur compounds. Figure 21.1 shows some logical steps leading to its production.

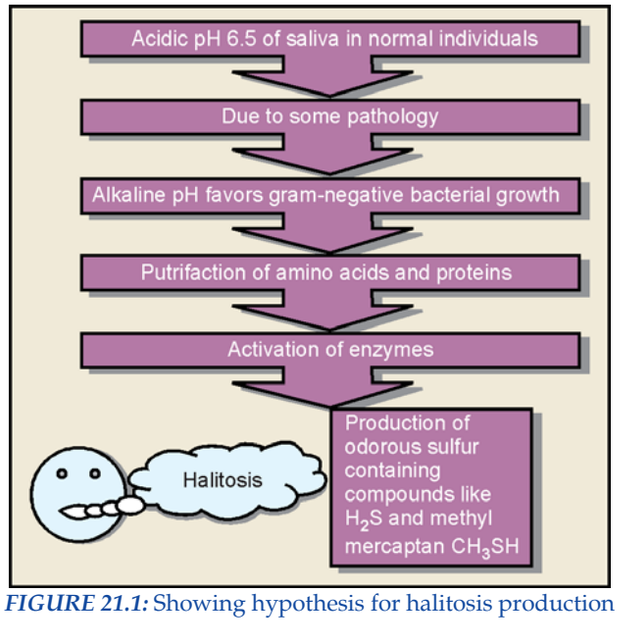

FIGURE 21.1: Showing hypothesis for halitosis production

Xerostomia

In Xerostomia, halitosis may be a clinical feature as there is lack of cleansing action of saliva. Normal saliva has a pH of approximately 6.5. The acidic pH suppresses the growth and proliferation of gram-negative bacteria and anaerobic bacteria. Alkaline pH favors gram-negative

bacteria and allows activation of enzymes required for putrefaction of amino acids whose end products are sulfur-containing compounds.

Breakdown of cellular proteins and amino acids to odorous sulfur containing compounds such as methyl-mercaptan (CH_3SH) and hydrogen sulfide (H_2S) results in halitosis. Other chemical compounds in the saliva causing halitosis are putrescine, cadaverine, histamine, indole and skatole.

MEASUREMENT OF HALITOSIS

Halitosis can be measured by using osmoscope, breath analyzers, high performance gas chromatography (HPGC) and mass spectrometric analysis of volatile sulfur compounds. In reality, odors are difficult to measure. A typical chromatogram of a patient with halitosis shows three peaks.

- Hydrogen sulphide is the highest
- Methyl mercaptan is the next highest, but it is the most objectionable and is smelly at lower concentrations.
- Dimethyl sulphide is the next peak, which is only a minor component.

Kozlousky A et al (1994)[10] investigated to test the association between BANA tests (Perioscan, Oral-B) and oral malodor parameters and found that BANA scores are associated with a component of oral malodor and that it could be used to measure volatile sulphides.

Indiscrimate use of mouthwashes may actually promote halitosis. Friedrish RE and Kristen U (1999)[7] assessed the adverse effects of mouthwashes by the pollen tube growth test (PTG test) and found the acute toxicity of mouthwashes exceeds the toxic effects of alcohol; if not used in correct amounts and number. In one study it was found that tongue was the major site for Volatile sulphur compounds (VSC) production.[8]

Zinc was found useful in control of many kinds of halitosis. Waler SM[9] (1997) tested the inhibitory property of zinc on production of VSC; he found that zinc has a valuable role in inhibiting odiferous VSC production.

Organoleptic Measurements

Organoleptic measurement is a sensory test scored on the basis of the examiner's perception of a subject's oral malodor. Although the objectivity and reproducibility of organoleptic measurement are poor, it does not require special equipment. It is the most practical procedure for evaluating a patient's oral malodor. Organoleptic measurement can be simply carried out by sniffing the patient's breath and scoring the level of malodor.

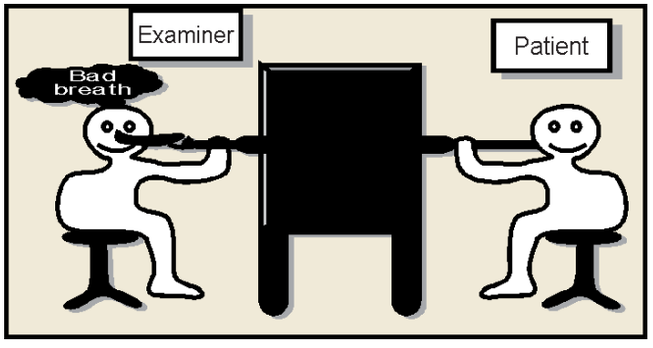

FIGURE 21.2: Organoleptic measurements

A straw or a plastic tube (24 mm in diameter and 10 mm in length) is inserted into patient's mouth. While the patient is exhaling slowly, the examiner judges the odor at the other end of the tube. In order to prevent the patient from seeing the examiner sniffing the tube, a privacy screen (50 cm × 70 cm) is used. The use of privacy screen allows the patient to believe that he or she is the subject of a specific malodor examination rather than the direct sniffing procedure (Fig. 21.2 and Table 21.1).[11]

Table 21.1: Organoleptic scoring scale[11]

Category	*Description*
0: Absence of odor	Odor cannot be detected.
1: Questional odor	Odor is detectable, although the examiner could not recognize it as malodor.
2: Slight malodor	Odor is deemed to exceed the threshold of malodor recognition.
3: Moderate malodor	Malodor is definitely detected.
4: Strong malodor	Strong malodor is detected, but can be tolerated by examiner.
5: Severe malodor	Overwhelming malodor is detected and cannot be tolerated by examiner

The sense of smell of the examiners must be standardized for organoleptic measurement of oral malodor. T and T Olfactometer™ an odor solution kit for measuring the olfactory sense, is utilized for this purpose.

Gas chromatography: Quantitative analysis of Volatile sulphur compound (VSC) by gas chromatography (GC) is considered to be gold standard for measuring oral malodor. Tonzetich and co-workers developed an auto injection system equipped with a 6-port-valve, a Teflon™ sample loop, and a Teflon™ column. This GC system allows for an accurate quantitative analysis of VAS in mouth air. VSC concentrations were calculated by a data handling software system (CHROMATOPAC C-R6A, Shimadzu, Kyoto, Japan) which was found to be compatible with the GC.[11]

Spoon test: The spoon test assesses the odor emanating from the dorsum of the posterior tongue. A plastic spoon is used to scrape and scoop material from the back region of the tongue dorsum. Five seconds later the spoon odor is evaluated at a distance of approximately 5 cm from the examiner's nose.[12]

Dental floss odor test: It is used to determine the presence of interdental plaque odor. Unwaxed floss is passed through interproximal contacts of the posterior teeth and the examiner assesses the odor by smelling the floss at a distance of approximately 3 cm.[12]

Saliva odor test: This test routinely involves having the subject expectorate approximately 1 to 2 ml of saliva into a Petri dish. The dish is covered immediately, incubated at 37º C for 5 min and then presented for odor evaluation at a distance of 4 cm from the examiner's nose.[12]

TREATMENT

Treatment must always be done by removing the most obvious causes in the oral cavity and step I through step V should be attempted gradually if the patient does not respond. The dentist must make a decision whether the problems lie in the oral cavity, or the other systems/ psychological roots. Once this medical treatment decision is made he must act accordingly (Fig. 21.3).

Nachnani S (1997)[4] suggested the use of commercial oral rinses an efficient treatment to control the halitosis problems. Overuse of mouthwashes can actually aggravate the problem.[7]

Frascella J et al (1998)[1] conducted tests on 12 male and female subjects to investigate the potential of chlorine dioxide mouthwash and found that a one-time use of chlorine-dioxide mouthwash improves mouth-odor pleasantness and reduces mouth odor intensity for at least 4 hours.

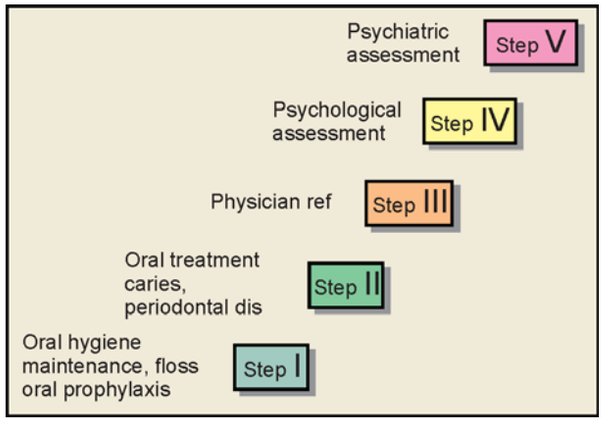

FIGURE 21.3: Diagram showing the steps in treatment needs of Halitosis. See text for details (Bailoor DN, Prasanna Kumar, Nillofer Shabnam 2004, Yenepoya Dental College and Hospital, Mangalore)

Niles HP et al[5] (1999) investigated the efficiency of a commercially available dentifrice containing Triclosan and a copolymer (Colgate total® toothpaste) for controlling long term i.e. Seven hour and over-night breath odor on 19 subjects and found that Colgate total toothpaste provides effective seven hour and over-night control of breath odor over other dentifrices.

Greenstein RB et al[2] (1997) investigated the antimalodor properties of oxidizing lozenges and found that only full-strength oxidizing lozenges significantly reduced tongue-dorsum malodor when compared to regular-strength oxidizing lozenges, chewing gums and breath mints.

Reingewirtz Y et al[6] (1999) studied 12 dental students in an open label explanatory study and found that chewing gum had a valuable mechanical role in cleaning dental surfaces and test gum may only temporarily control bad breath.

SUMMARY

A. Identification of the cause oral, medical or psychological should guide the dentist into proper referral.
B. Patients should be instructed to maintain proper oral hygiene, using water pick, electrical toothbrushes and good foaming tooth pastes.

C. Different agents have significantly been effective in reducing malodor:
- Mouthwash containing chlorine dioxide
- Oxidizing lozenges on high-strength.
- Toothpaste and mouthrinses which has pure solvent-glycerol (GLY) and polyethylene (PEG) with triclosan dissolved in it.
- Chewing gums preferably with zinc component in it.
- Use of flavoured mouthrinses can be valuable in reducing halitosis but only for temporary relief.
- Use of **antibiotic mouthrinses are not advisable** as they can suppress normal microflora which has a protective role by preventing growth and multiplication of the disease-causing strains.
- Also mouthrinses containing cysteine should be avoided since it can produce volatile sulphur compounds that are the main cause of halitosis.
- Mouthrinses should be taken according to the instructions given about the number and quantity. Because if not used in proper amounts and intervals, it can have adverse effects like stomatitis and desquamation of oral mucosa.
- Dryness of mouth should be managed by using drugs, e.g.Pilocarpine which stimulate saliva flow or by using artificial saliva substitutes.
- But drugs with iodine, chloral hydrate and also antineoplastic drugs should be avoided.

CONCLUSION

The Dental surgeon must assess if the patient needs dental care, medical care or psychological counseling in the chronic case of Halitosis. Sometimes two of the three causes may be culprits. Use of different pastes, mouthwashes or over the counter products can give only temporary relief if proper diagnosis is not made.

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22 Dysphagia

INTRODUCTION

The difficulty in swallowing brings the patient to dental surgeon because most laymen believe that only oral cavity takes part in the swallowing process. The truth is that swallow consists of the oral phase, the pharyngeal phase and the esophageal phase. The esophagus appears to be a simple organ, and yet the esophageal diseases are common. They range from heartburn, aspiration, obstruction and hemorrhage.

NORMAL SWALLOW PHYSIOLOGY

Swallowing or deglutition is a reflex response controlled via vagus Xth nerve and its center is located in the medulla oblongata.

It is divided into 3 stages:

Stage I—Oral stage	→ Voluntary stage
Stage II—Pharyngeal stage	→ Involuntary (reflex) stage
Stage III—Esophageal stage	→ Involuntary (reflex) stage

The oral stage involves chewing food and forming it into an oral bolus while propelling it by the tongue into the posterior pharynx.

In the pharyngeal stage, food is passed from the pharynx across the upper esophageal sphincter (UES) into the proximal esophagus.

The entire process occurs in 1 second and involves 5 important steps:

1. The soft palate is elevated and retracted to prevent nasopharyngeal regurgitation.
2. The vocal cords are closed and the epiglottis swings backwards to close the larynx and prevent aspiration.
3. The UES relaxes.
4. The larynx is pulled upward, thereby stretching the opening of the esophagus and upper sphincter.
5. Contractions of the pharyngeal muscles provide a driving force to propel food into the esophagus.

In the esophageal stage ingested food is transported from the UES to the stomach while the lower esophageal sphincter (LES) is relaxed. This is accomplished primarily by an orderly stippling wave initiated by swallowing and progressing along the esophagus (i.e. primary peristalsis).

After the food bolus passes, the LES re-establishes a tonic contraction, thereby preventing regurgitation of gastric contents.

DEFINITION

Dysphagia or difficulty in swallowing denotes the subjective sensation evoked when a food bolus is impeded in its passage from the mouth down the esophagus, to the stomach. Stoschus B and Allescher HD[1] state " Dysphagia describes the disability or problems in swallowing a wet

or dry bolus properly and is normally associated with an impaired transport of bolus. It is accompanied by pain sensation in the chest mostly caused by impaction of the food bolus in the esophagus".

Kim CH et al (1996)[2] has classified dysphagia as—

I. ***Obstructive***: When an obstructive lesion was present. It is detected on esophagoscopy or barium swallow,
II. ***Motility related***: When abnormal motility is shown on esophageal manometry in the presence of normal esophagoscopy or barium swallow,
III. ***Nonmotility related***: When manometry to esophagoscopy or barium swallow was normal" (Probably suspecting psychological causes here).

Helpful questions to ask patients with dysphagia:

1. Is the difficulty in swallowing a problem in initially swallowing or does the food stick going down after its swallowed?
2. What kind of foods is difficult to swallow: liquids or solids?
3. Is the dysphagia intermitted or is it getting progressively worse?
4. Where does the food get stock going down?
5. Are there associated symptoms such as chest pain, heartburn, pain with swallowing, regurgitation or a constant feeling of lump in the throat?
6. Are there associated symptoms such as hoarseness or coughing and choking while eating?
7. Are there associated neuromuscular symptoms such as double vision, facial troop, and change in voice, muscle weakness, difficulty in walking or using hands to hold things?

   Buchholz DW (1994)[3,4] state that "Causes of neurogenic dysphagia include stroke, head trauma, Parkinson's disease, motor neuron disease and myopathy".
8. Has there been any weight-loss in the last several months?
9. Are there associated medical conditions such as scleroderma, diabetes, stroke, or arthritis
10. What medications are you currently taking, including over the counter medications? (Includes the medicines stopped recently but taken for some time).

CLINICAL FEATURES

Usually symptoms such as painful swallowing, retrosternal pain are seen. It may be difficult to differentiate esophageal and cardiac pain and there's evidence that gastroesophageal reflux may lower the threshold for angina.

Regurgitation of gastric juice into the lower esophagus and the back of the mouth, cause heartburn due to incompetent lower esophageal sphincter.

If food is accumulated in the esophagus: nausea, vomiting, weight loss is common.

EVALUATION OF PATIENTS WITH DYSPHAGIA

History is the most important contribution to diagnosis as it can give important clues regarding the cause of dysphagia (Fig. 22.1 and Table 22.1).

Algorithm for the Differential Diagnosis of Dysphagia

1. *Oropharyngeal dysphagia* is usually described as the inability to initiate the act of swallowing. It is a "transfer" problem of impaired ability to move food from the mouth into the upper esophagus. Cause of oropharyngeal dysphagia is cerebrovascular accidents, oropharyngeal structural lesions, systematic and local muscular diseases, neurologic disorders.
2. *Esophageal dysphagia* results from difficulty in "transporting" food down the esophagus and may be caused by motility disorders or mechanical obstructing lesions. It is caused due to neuromuscular disorders, mortality abnormalities and intrinsic and extrinsic obstructive lesion.[5.]

Abnormal physical signs are unusual in patients with esophageal disease, but epigastric tenderness may indicate *patients complaint any one of the following:*

- Esophagitis
- Peptic ulceration
- Carcinoma

In case of esophageal carcinoma we can observe an epigastric mass and palpable supraclavicular lymph nodes.

Table 22.1: Causes of dysphagia

Oropharyngeal dysphagia	*Esophageal dysphagia*
Neuromuscular disease *Diseases of the central nervous system* • Cerebrovascular accident • Parkinson's disease • Brainstem tumors • Degenerative diseases • Amyotrophic lateral sclerosis *Multiple sclerosis* • Huntington's disease • Postinfectious • Poliomyelitis • Syphilis *Peripheral nervous system* • Peripheral neuropathy *Motor endplate dysfunction* • Myasthenia gravis	**Neuromuscular disorders** • Achalasia • Spastic motor disorders • Diffuse esophageal spasm • Hypertensive lower esophageal sphincter
Skeletal muscle disease (myopathies) • Polymyositis • Dermatomyositis • Muscular dystrophy (myotonic dystrophy, oculopharyngeal dystrophy) • Cricopharyngeal (upper esophageal sphincter), achalasia	• Vascular compression • Enlarged aorta or left atrium • Aberrant vessels • Mediastinal masses • Lymphadenopathy • Substernal thyroid
Obstructive lesions *Intrensic structural lesions* • Tumors • Inflammatory masses • Trauma/surgical resection • Zenker's diverticulum • Esophageal webs *Extrinsic structural lesions* • Anterior mediastinal masses • Cervical spondylosis	• Tumors • Strictures • Lower esophageal rings (Schatzki's ring) • Esophageal webs • Foreign bodies

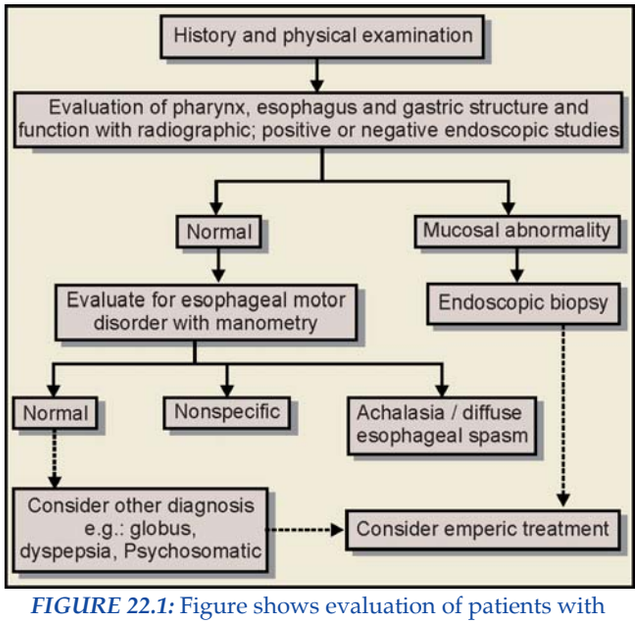

FIGURE 22.1: Figure shows evaluation of patients with dysphagia

Initially there is difficulty in swallowing solid foods, then progresses to difficulty in swallowing liquids –

- Anorexia, weight loss
- Regurgitation, aspiration
- Pneumonia may occur with tracheoesophageal fistula
- Substernal pain
- Hoarseness due to impingement on recurrent laryngeal nerve
- Melena and gastrointestinal bleeding.

Determining the type of bolus and the temporal progression of dysphagia may help differentiate between an obstructive lesion and a motor disorder.

In neuromuscular disorder—The patient notes dysphagia for both solids and liquids.

A neurological examination should be performed to look for evidence of neuromuscular disorder, such as a prior CVA or Parkinson's disease.[6] The graphic description

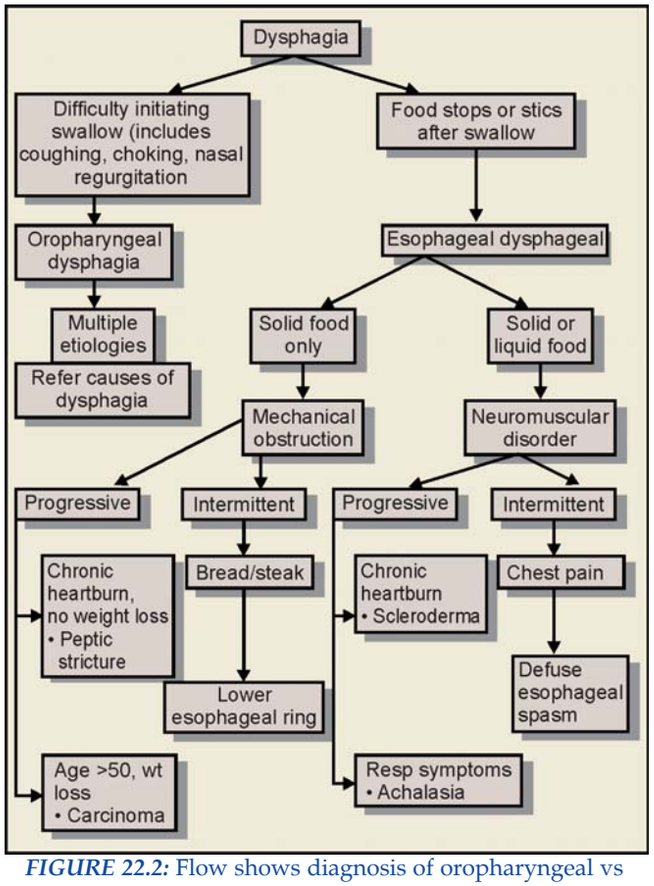

FIGURE 22.2: Flow shows diagnosis of oropharyngeal vs esophageal dysphagia

by Buchholz DW [3] 1994 which says " Drooling, difficulty in initiating swallow, nasal regurgitation, difficulty managing secretions, choking, cough episodes, food sticking in the throat" all these should alert the dentist into knowing that a neurologists opinion is a must.

- The patients with nasopharyngeal dysfunction and the pharyngoesophageal (Zenker's diverticulum) dysfunction.
- Patients with achalasia describe an obstruction in the lower sternal area. There is decrease esophageal clearance of solids and fluids and may cause pulmonary aspiration.
- Episodic dysphagia for solids may be produced by an esophageal web or ring without symptoms or weight loss.
- Autoimmune sialoadenitis is frequently associated with dysphagia. Various studies including that of Anselmino M et al [7] 1997 who states that about one third of patients with primary Sjögren's syndrome have an abnormal esophageal peristalsis that is responsible for dysphagia, whereas decreased salivary outflow exacerbates the swallowing discomfort".
- Many medications precipitate dysphagia. These include tetracycline, doxycycline, minocycline, quinine, aspirin. Here acute development of retrosternal pain is observed, usually exacerbated by swallowing (odynophagia).
- Immunosuppressive drugs used in cancer chemotherapy may precipitate the fungal esophagitis, which may present as dysphagia.
- Drug reactions like erythema multiforme or Stevens Johnson syndrome can also cause desquamation and ulceration up to the level of esophagus causing the dysphagia (Fig. 22.2).

DIAGNOSTIC TESTS

1. ***Barium swallow or barium meal*** is the first evaluation indicated. Radiographic evaluation shows extrinsic structural lesions, e.g. thymoma, initiative obstructive lesions, e.g. esophageal rings and webs.
2. ***Indirect laryngoscopy***.
3. ***Endoscopic examination*** and use of special endoscopes for esophageal biopsy.
4. ***Video fluoroscopic evaluation***: This is helpful for evaluation of swallowing function and slow motion replay of the complex events during swallowing.

   Brian C (1998)[8] vouches for video fluoroscopy as the most sensitive technique in identifying oropharyngeal alterations of swallowing.
5. ***Electromyographic*** studies coupled with blood screens help in neurogenic dysphagia evaluations.
6. ***MRI***—Magnetic resonance imaging is also definitely one of the tests to consider when the cause remains undetected.
7. ***Manometry of esophagus*:** Determining the force or pressure of swallow using a esophageal manometer lends a clue to those perplexing cases.
8. ***Esophageal scintigraphy:*** Useful screening test for esophageal motility disorders. It determines functional

obstruction and shows any abnormal transit of the radionuclide bolus.

TREATMENT

Based on the multidisciplinary discussion with physician, neurologist and the psychologist, the oral medicine specialist usually decides one of the following:

1. All the oral causes of dysphagia are tackled before referring the patient to the other specialists.
2. Nutritional regimen of balanced vitamins and minerals together some form of laxatives since the low fiber food is more easily swallowed. Nutritionists ultimately may decide on exact nature of food texture that makes the patient comfortable. Nutritionist is definitely one of the team members here.
3. The need for surgery, regular or endoscopic must be made consulting with gastroenterologist.
4. Exercise programs aimed at improving the neuro-muscular control is indicated.
5. Intraoral prostheses may be designed for patients who have undergone cancer surgery to facilitate swallowing.
6. Use of artificial saliva and other lubricants may make the patients swallow much comfortable.

CONCLUSION

The description of difficult swallowing by a patient should alert the dentist. After removal of the oral causes if the problem persists then the possibility of referral to a specialty medical center should be considered. Since carcinoma of the esophagus is a real possibility in many cases gastroenterologists opinion is the first to be sought and the oral medicine and radiology specialist must work very closely with this professional to chalk out the diagnostic tests required. In advanced cases, involving nutritionist and a clinical psychologist helps in a more complete health care management of the problems that are difficult to swallow!

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23 Radiation Physics, Properties and Production of X-rays

INTRODUCTION

Our wonderful universe is made up of two main types of stuff. Matter, which is made up of atoms and molecules and radiation energy, which rapidly travels from one place to another till it, dissipates. Matter and energy are inter-convertible, Professor Albert Einstein taught this fact for the first time.

NATURE OF RADIATION

Physicists tell us that radiation has a dual nature. Sometimes it behaves like packets of energy called as photons and sometimes it behaves like a continuous spectrum of electrical and magnetic fields traveling through space. Basically the radiation is transfer of pure energy from point A to point B in space. The amount of energy in the electromagnetic (EM) waves is dependent on its frequency, higher the frequency more the energy.

The energy of an EM wave is given by the formula

$$E=P \times F$$

E = Energy of the radiation in kilo electron volt keV
P = Planck's constant (6.25×10^{34} joule seconds)
F = Frequency (Hertz)

The wavelength of a beam varies inversely as the frequency. This means that greater is the wavelength lesser will be its energy.

The radiation is of two types—the particulate and the waveform pure energy. The particulate radiation consists atomic nuclei or sub-atomic particles moving at high speeds examples of these are—the alpha particles, the beta particles and the cathode rays. The alpha particles constitute of two protons and two neutrons and are slow moving and easily absorbed even by a thin paper. The beta particles and cathode rays are both high-speed electrons. The gamma radiation on the other hand is pure electromagnetic radiation that is indistinguishable from the X-rays. The only difference being that gamma rays emanate from the radioactive nuclei and X-rays from the braking radiation in dental tube.

ELECTROMAGNETIC SPECTRUM (EM SPECTRUM)

What we are able to see, or our use of eyes is helped by narrow band of radiation termed as visible spectrum made up of VIBGYOR or violet, indigo, blue, green, yellow, orange and red colors. The real band of electromagnetic spectrum actually goes far below (in frequency) infrared and far above (ultraviolet) radiation. The photons used in dental radiography come in the range of 0.1 to 0.001 nm wavelength (Figs 23.1 and 23.2).

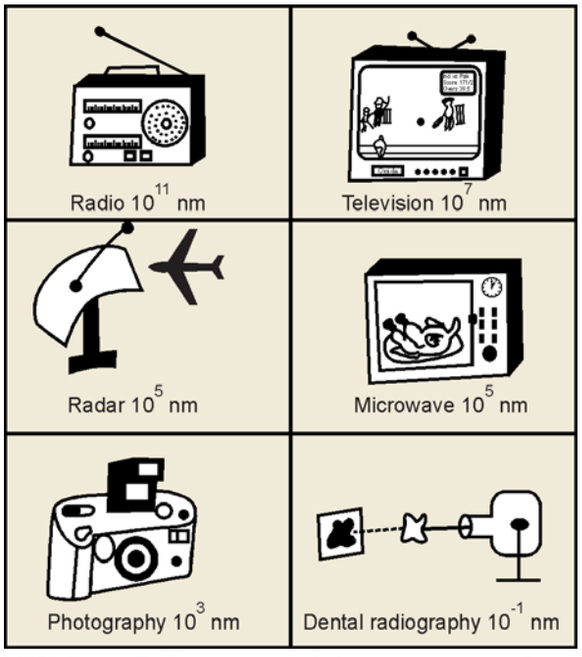


FIGURE 23.1: Different types of electromagnetic radiation

Two Main Types of Medical Uses of X-rays

The two types of X-rays used in health sciences can be classified as *Diagnostic* and *Therapeutic*. The diagnostic X-rays are used to make the shadow pictures of teeth, face and the jaws. This helps us to look at calcified structures and evaluate their insides.

The therapeutic X-rays are usually very powerful radiation beams coming from radioactive nuclides, cyclotrons or linear accelerators. They help us to literally burn away the malignancies of the jaws. The branch of oncology which deals with the use of therapeutic radiation is called as radiotherapy.

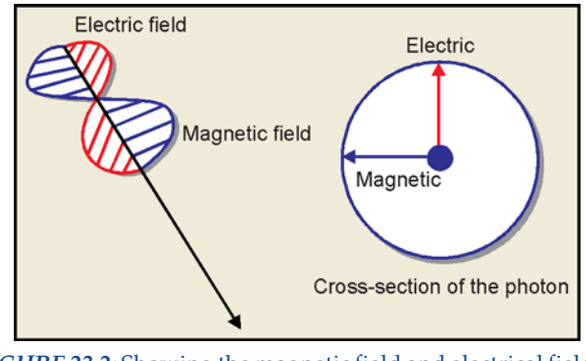


FIGURE 23.2: Showing the magnetic field and electrical field at right angles to the direction of passage of the photon. In the cross-section we can appreciate that the X-ray photon is coming out of the page and the two field s are at right angles to each other

PRODUCTION OF DIAGNOSTIC X-RAYS

Diagnostic radiation for seeing the dental and facial structures can be produced in two ways:

1. Use of radioactive isotopes for diagnostic purposes.
2. Use of 'Bremsstrahlung' interaction to generate the X-ray radiation.

1. Use of radioactive isotopes with exposure facilities to pump them in appropriate chamber to expose the radiographic film. Such as in the forensic radiology or in dental radiology in far flung sites with no electrical connections.

   Kircos LT et al[1] 1986 developed a portable radiographic X-ray camera made of tantalum and aluminum. Radioactive iodine (^{125}I), ytterbium (^{169}Yb), and gadolinium (^{153}Gd) have been used together with Kodak fast screen films to reduce the amount of exposure. Some of the researchers have used a Polaroid type dental film, which needs no processing, and as soon as it is stripped and waved in air it will show the image. They have enthusiastically endorsed this unit saying that it is lightweight, portable and completely safe for operator and patient. It can be operated with ease in deepest jungles or in war zones where soldiers may be fighting.
2. Use of Bremsstrahlung type of interaction, which involves the braking, or stopping of high-speed electrons. The kinetic energy of these electrons is partially converted into X-ray radiation. Let us now see in detail how the X-rays are produced in a typical dental X-ray tube.

Circuits of the X-ray Tube

The energy for the X-ray tube comes from two circuits; the high voltage circuit and the low voltage circuit.

The low voltage circuit energizes the filament of the cathode and helps to create a electron cloud by process termed as 'Thermionic emission'.

The high voltage circuit creates a potential difference between the cathode and the anode and helps to accelerate the electrons jumping in the cloud, focused by molybdenum cup.

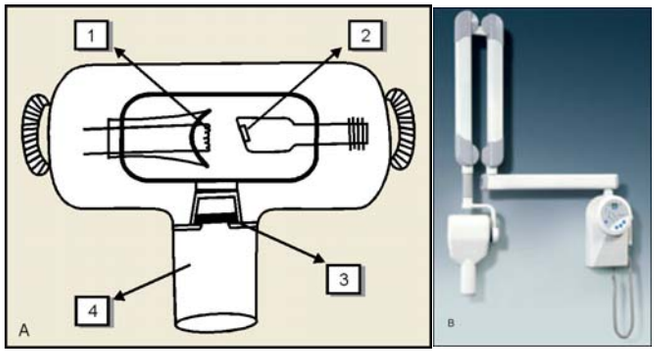


FIGURE 23.3: Showing the structure of the X-ray tube with basic components of cathode(1), anode(2), aluminium filter (3) and collimator(4).

Structure

The dental X-ray tube consists of the cathode (negatively charged), the anode (positively charged) and the intervening vacuum in the tube (Fig. 23.3).

CATHODE consists of a filament and focusing cup. The coil of tungsten wire, which forms the filament, is 0.2 cm in diameter and slightly less than 1 cm in length. This coil is recessed in a molybdenum-focusing cup.

ANODE consists of a copper stem and a tungsten target. The surface of the copper stem is 20° to the perpendicular in which the tungsten target is embedded. Tungsten is chosen because of its high melting point, high atomic number, low vapour pressure in vacuum. Copper is a good conductor of heat and is strong enough to withstand the onslaught of the high speed electrons. The rapid dissipation of heat by copper helps to keep target temperatures at manageable levels.

PROCESS

This tube is lodged in two types of circuits, the low voltage circuit and the high voltage circuit. The low voltage circuit (10 volts) is applied across the cathode filament, which heats it. The hot filament emits an electron cloud by a process termed as "*thermionic emission*" (Fig. 23.4). This cloud is focused into a small area by the negatively charged molybdenum cup. The high voltage circuit (65,000 to 90,000 volts) is always applied after the electron cloud is ready for dispersal. The high voltage accelerates the electrons to the tungsten target. The focusing cup helps to focus the high velocity electrons to the target. The vacuum serves two purposes it helps the high speed electrons to hit the target without undergoing collisions of the air molecules and the heat generated in the tungsten coil does not burn up.

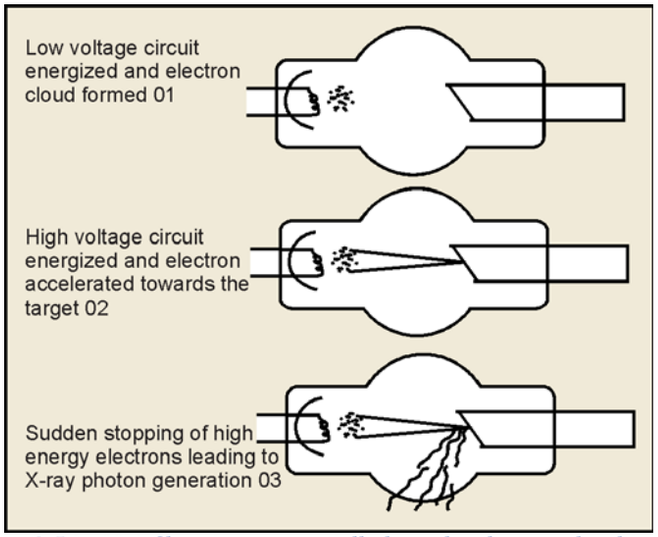


FIGURE 23.4: Showing sequentially how the electron cloud is generated, accelerated and converted into heat and X-ray photon energy at the target of anode

The point at which the high speed electrons hit the tungsten target is called the focal spot. At this point the kinetic energy of these electrons is converted into 99% heat and 1% X-ray radiation, which is fanned out around in the tube and emanates from the window provided for it.

The sharpness of the radiographic image increases as the size of focal spot decreases. But since 99% of the energy is converted to heat, the spot designers cannot make it too small. The ingenious way of making the focal spot apparently small without actually making it small is termed as the ***LINE FOCUS PRINCIPLE***. The face of the anode in which the target is recessed is slanted at an angle of 20°. This means that a target, which is 1 mm × 3 mm in size, appears to 1 mm × 1 mm when viewed at the direction in which the X-rays are generated (Fig. 23.4).

Transformers: These electrical machines help in transforming the voltage from street current to low voltage (step down transformer); and from street current to high voltage (step up transformer). Auto-transformer is a complex electronic equipment which helps to control the voltages across the circuits as required by the

radiographers, in an oversimplified kind of explanation it is step up and step down transformer rolled in one.

PRODUCTION OF X-RAYS: The kinetic energy of the electron stream results in the generation of the photons of X-rays. The underlying mechanisms are termed as Bremsstrahlung and characteristic radiation (Fig. 23.5).

Bremsstrahlung radiation results from the:

a. Direct hit of the electron on the nucleus of the tungsten target.
b. Passage of electrons close to the nucleus resulting in its loss of energy.

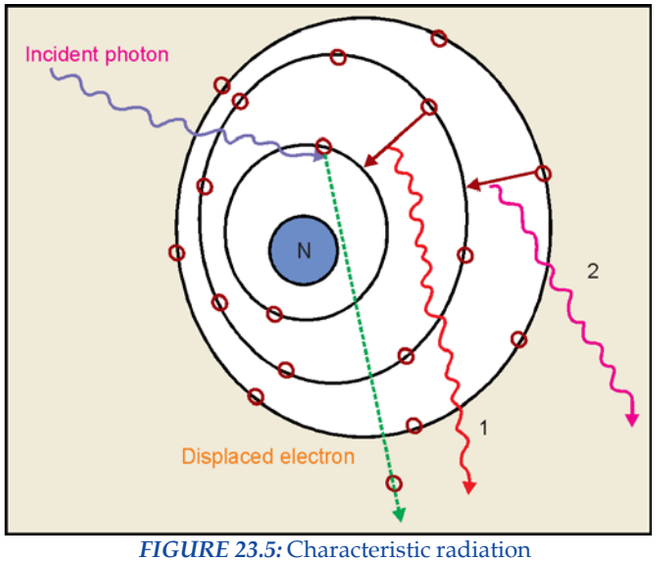


FIGURE 23.5: Characteristic radiation

Characteristic radiation results, when, the high speed electron knocks off the inner orbital electron of tungsten target, resulting in a cascading of electrons from the higher energy levels resulting is multiple daughter photons of radiation being generated.

Most of the photons are generated by Bremsstrahlung process in dental tube (Fig. 23.6).

Factors Controlling the X-ray Beam Emanating from the Dental Tube

1. **Product of tube current (mA) and the exposure time(s)** (mA × s)—the multiplication factor of milliamperage and the exposure in seconds. Greater the mAs darker will be the film. Clinicians use the term called as *density* of the film for this darkening. The quantity of the electrons is controlled by the mAs multiplication factor.

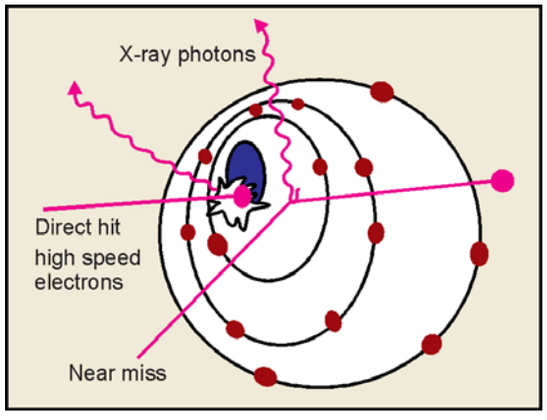


FIGURE 23.6: Showing electrons from the cathode undergoing direct hit or near miss and in both cases generating X-ray photons of differing energies.

2. **Tube voltage (kVp-translated kilo voltage peak)**—The voltage increases the penetration (kinetic energy) of the X-ray photons. The number of photons generated is also increased as the voltage increase; the mean energy of the beam and the maximal energy of individual photons appear to be more.
3. **Filtration**—the primary beam generated is of polychromatic variety or of different frequencies and energies. The low energy X-rays will not reach the film and will be absorbed by the skin and teeth, hence they need to be removed from the main beam. This is achieved by filtration using aluminum filters (Fig. 23.7). Here three terms are to be understood ***inherent filtration***- the filtration that is in the dental tube due to glass and heavy oil (0.5 mm Aluminum (Al) in the dental tube). The ***added filtration*** which is usually 1 mm of Al and ***total filtration*** which is the sum of the two filtrations which is about 1.5 mm of Al.

   1.5 Aluminium discs perform filtration at 65 kVp and 2.5 mm of aluminium would be required for kVp upto 90 kVp. These discs are inserted by manufacturers at the base of the cone.

   After filtration the only beam that is allowed to emanate is the high energy beam, which will be of great diagnostic value since it helps in shadow casting of the hard-calcified structures in the orofacial regions.
4. **Collimation**—means shaping of the X-ray beam such that no unnecessary exposure is done to skin and tissues at entry areas. This is achieved using the lead cylinders of 7 cm in diameters or the lead lined

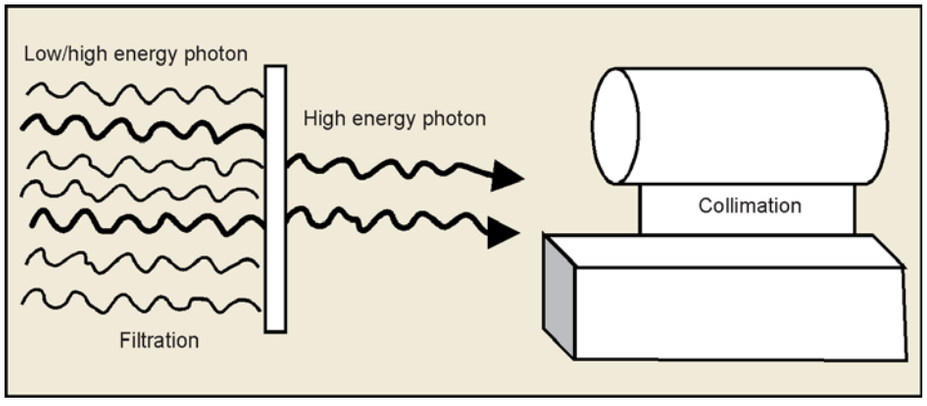


FIGURE 23.7: Figure depicting filtration and collimation

rectangular shapers. Collimation reduces the Compton's scatter generated, which decreases the fogging and increases the clarity.

5. **Distance**—greater is the distance between the tube and the patient, lesser is the intensity of the X-rays due to spreading out. The distances are actually calculated from target to the film. The intensity follows the inverse square law. The intensity of the beam is inversely proportional to the square of the distance from the target to the imaging media.
   Formula:
   $$I_1/I_2 = (D_2)^2/(D_1)^2$$
   Where I_1 and I_2 are intensities of X-ray photons
   And D_1, D_2 are the respective distances from the target.
6. **Intensifying screens**—use of regular and rare earth intensifying screens result in the need for reduced exposure of X-rays to patient. The radiographer needs to reduce the factors associated with the X-ray beam mentioned above in order to compensate for the increased sensitivity of the film, screen combinations.
7. **Grids** need the mAs and kVp to be increased to a certain extent so that the image obtained is sharper and better. Hence, the radiographer needs to increase the factor no. 1 and 2 so that optimal image is obtained.

Interaction of X-rays with Matter

When a traveling photon encounters matter two things can happen:

It may pass clean through without any interaction at that moment, or

It may undergo one of the three types of interactions mentioned below:

i. **Classic or coherent scattering:** When a X-ray photon of low frequency interacts with low energy electron of outermost orbit of the interacting matter this photon is completely absorbed by the electron. It starts vibrating with a higher energy state, which obviously is unstable. In a brief interval (almost instantaneous) a new photon is ejected in a different direction of the same energy as the incoming X-ray photon. The observer would in fact feel that the X-ray photon came in, interacted and came out in a different direction altogether. **8-10%** of the interactions of this type occur (Fig. 23.9A).

ii. **Compton scattering:** A slightly higher energy X-ray photon interacts with the outer electron of the matter.

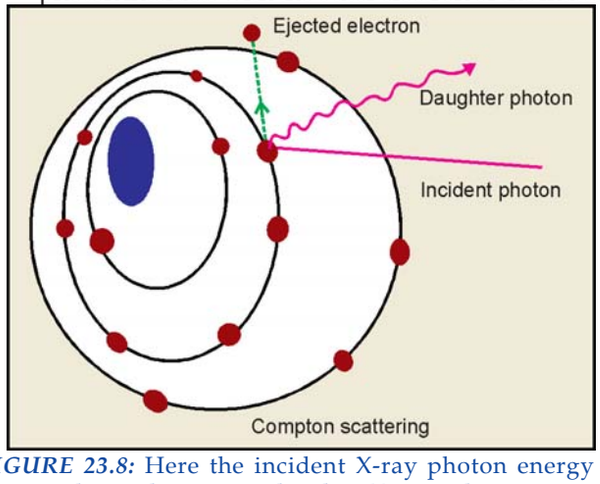
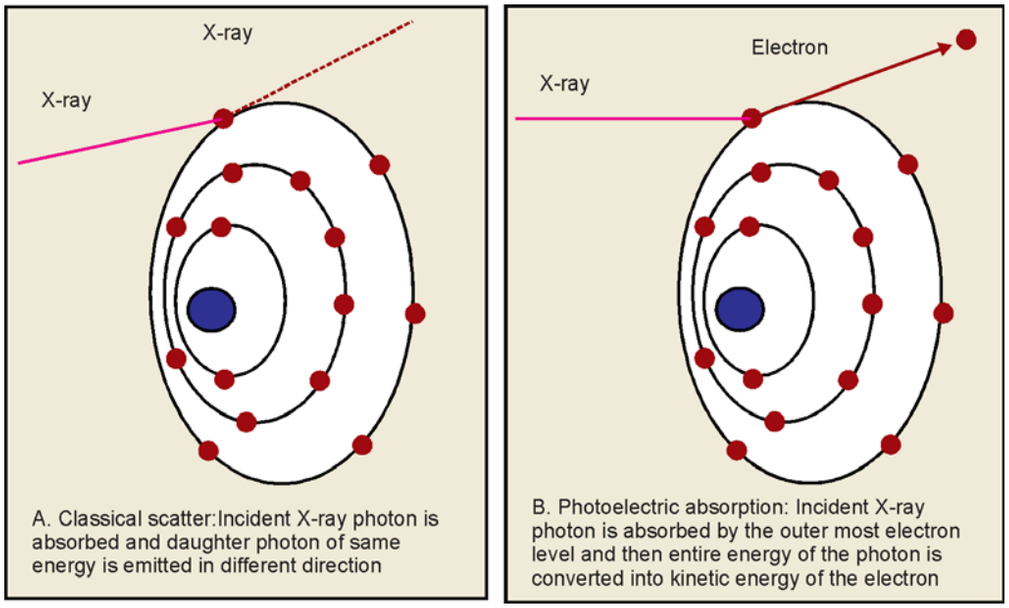


FIGURE 23.8: Here the incident X-ray photon energy is converted into a low energy daughter X-ray and one energized electron which is ejected



FIGURES 23.9A and B: Shows the interaction of X-rays with matter. A depicts classical scatter and B depicts photoelectric absorption. Nillofer Shabnam, Prassanna Kumar, Bailoor DN 2004

The electron absorbs a part of the kinetic energy of the beam and goes off in one direction; this is the recoil electron. The remaining energy is deflected in some other direction as a low energy photon. In dental X-ray beam about **60-65%** of the photons interact with facial tissues by Compton scattering (Fig. 23.8).

iii. **Photoelectric absorption:** When a high energy X-ray photon hits a electron of the inner shell, say the K shell, then the electron takes the energy of the X-ray and gets ejected. This is termed as the photoelectron. This leaves the atom in an ionized state, and gradually the electrons from higher energy levels tend to cascade down to lower levels. The energy differences in the orbital level are ejected as characteristic radiation. Depending on the energy of the original radiation one or many daughter photons will be given out. Finally only the outer most shell has deficiency. This atom is still in ionized state and has to pick up electron from the environment around to become stable once again. 25-30% of the photons in dental radiology range undergo photoelectric absorption (Fig. 23.9B).

Classical scattering—the incident X-ray photon and the one emitted out have the same energy and no change ultimately occurs in the atom interacting with this photon. The direction of the daughter photon is different from that of incident photon.

Fate of secondary electrons—The electrons that are emitted by any of the above mentioned interactions give up their energies in two ways. One by creating ionization and second by giving out low energy daughter X-rays. Ultimately both are dissipated into the surrounding matter in form of heat.

Half Value Layer (HVL)

The HVL denotes the penetrating quality of the X-ray beam that one may be trying to quantify. It is defined as the thickness of the absorber, which will reduce the number of photons exactly into half, e.g. we tend to use a dental X-ray beam which is 1.5 mm HVL quality for upto 75 kVp. The higher kVp beams like 90 kV will tend to have 2.5 mm HVL quality.

SUMMARY

The basic understanding of the atomic structure and the tube design helps the dental surgeon in the understanding the various properties of X-rays emanating from the tube and how to take good care of the X-ray unit.

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24 Radiation Hygiene

INTRODUCTION

Today due to a general increase in the health care awareness the patients are asking in the dental clinic whether X-rays are safe? More so when the children and pregnant women are being treated. Here the dentist must give a rational and scientific answer and not shrug off the question as being naïve. The dentist must try to explain concepts of naturally occurring radiation and that the diagnostic dental radiation most of the times is less in dose or equal to that of background radiation. The dentist must explain to the patient that the equipment used in diagnostic dentistry today is of highest quality and confirms with the radiation protection rules laid down by BARC (Bhabha Atomic Research Center, Mumbai—Radiation protection division and other Indian Governmental Agencies) and hence the well collimated and correctly filtered X-ray beam will not be a health hazard in hands of a qualified dental professional.

Radiation hygiene includes understanding the risks of ionizing radiations and planning the steps to reduce its adverse effects to the minimum or ALARA, i.e. **A**s **L**ow **A**s **R**easonably **A**chievable, by use of quality assurance method in a clinical setting.

DOSIMETRY

Determining the quantity of the radiation energy is termed as dosimetry. It could be placed dosimetry or it could be personnel dosimetry.

Units of interest to the dental surgeon –

Unit traditional	*SI*	*What it measures*	*Conversion*
Roentgen (R)	Coulomb/kg	Exposure	1C/kg = 3876R
Rad	Gray	Absorbed dose	1Gy = 100 rads
Rem	Sievert (Sv)	Dose equivalent	1Sv = 100 rems

RADIATION BIOLOGY

The science of the effects of the radiation on living organisms is called radiation biology. In this chapter, we discuss these effects as they apply to biological molecules and cells of the body. We also discuss how much radiation is received from dental radiographs, the risks that are involved with these exposures, and how to protect yourself and your patient from unnecessary or excess radiation.

As a beam of radiation passes through matter, it gradually weakens and eventually disappears. The energy of the beam is transferred to the material through which it passes. This transfer of energy is called **absorption.**

- **Direct effects:** There are several ways for an X-ray photon to interact directly with the atoms in the material through which it passes. If the material is living tissue, the absorption of energy from an X-ray beam may result in chemical (molecular) changes. These **direct effects** of radiation include

- Breaking molecules in to smaller pieces
- Disrupting molecular bonds
- Forming new bonds within molecules
- Forming new bonds between molecules.

- **Indirect effects:** Molecules not directly affected by X-rays can also be altered. Living organisms consist mostly of water. If an X-ray photon interacts with water and oxygen, charged atoms called free radicals are formed. The process is called **radiolysis**. A free radical readily reacts with other biological molecules. Radicals may remove electrons or hydrogen atoms from organic molecules, add bonds, or initiate between molecule bonding.

  Examples of radicals created by the radiolysis of water include

  X-ray photon + $H_2O \rightarrow H + OH$ (hydrogen + hydroxy 1 radical)

  $H + O_2 = HO_2$ (peroxy 1 radical)

  In addition, two hydroxy 1 radicals (OH ×) can combine to from hydrogen peroxide (H_2O_2), a chemical toxic to most cells.

  The effects caused by the radicals are not directly the result of a molecule being "hit" by radiation but because the damage is mediated by a free radical, it is referred to as an indirect effect of radiation. The indirect actions of X-rays can damage biological molecules as easily as the direct actions. Any changes in an organic molecule, no matter how it is damaged, may result in altered cell function. In summary then, **indirect effects** of radiation include:
  - The production of free radicals, which in turn cause
  - The alteration of other molecules.
- **Cellular effects:**[5] A cell has two basic components, the nucleus and the cytoplasm. Ionizing radiation may affect either area, or both. Damage in the nucleus often affects the chromosomes. As you remember, the chromosomes contain DNA (deoxyribonucleic acid). The DNA in each organism is composed of a particular series of bases. The specific order of the DNA bases is the foundation for the genetic code, which makes each organism unique. Radiation may alter the base sequence of the DNA molecule and make it defective.[4]

  Defective DNA may lead to the disruption of the mechanisms for cell division (mitosis). There may be delayed cell division or loss or reproductive capacity of the cells. Errors that are permanently incorporated into the DNA are passed on to future generations of those cells as **mutations**. If the defective DNA happened to be contained in a reproductive cell (sperm or ovum), then the defect may be passed along to future generations of organisms. This, then, would be a **genetic effect** of radiation.

  Radiation can also affect cellular cytoplasm. Cell may develop the following problems if the cytoplasm is damaged:[6]
  - Increased permeability or rupture of membranes
  - Rendering organs such as lysosomes, endoplasmic reticulum, and mitochondria, nonfunctional
  - Inactivation of enzymes
  - Coagulation of the cytoplasmic fluid.

  Any one of these changes could result in disruption of cell function or even cell death.

  All body tissues except the reproductive cell are called somatic tissues. The somatic effects of radiation may occur in the cell cytoplasm or nucleus. If the damage to the cells is severe enough, the organism may become ill or even die. However, somatic effects of radiation are not passed along to future generations as are genetic effects. Genetic effects occur only in the reproductive cells.
- **Cellular sensitivity to radiation:** Some cells are more sensitive to radiation than others. A cell will be more sensitive to radiation if it has any of the following characteristics:
  - A high mitotic rate (undergoes frequent cell division)
  - A long mitotic history (undergoes many divisions over time)
  - A primitive or immature nature (must undergo further growth or development)
  - An undifferentiated nature (is not highly specialized).

  An exception is the lymphocyte, which is a highly specialized cell of the immune system. It will not divide once it is mature. However, the **small lymphocyte** is probably the **cell most sensitive to radiation.**
- **Short and Long-term effects of radiation:** Harmful effects of radiation do not show up immediately. There is a time lag between exposure to radiation and the

signs and symptoms of biological damage. This period of time is called the **latent period,** which may be as short as a few hours or as long as twenty years or more. The length of the latent period depends on the total dose of radiation received and the amount of time it took to receive that dose. Higher the dose and the quicker the rate, shorter will latent period be.

- **Short-term** or acute effects of radiation usually result from high doses to the entire body. Symptoms may include nausea, vomiting, diarrhea, fever, loss of hair, hemorrhage, and even total body collapse. Long-term or chronic effects of radiation are usually due to doses of radiation received over a long period of time.

  There are **cumulative** effects of repeated radiation exposure. Tissues do have the capacity to repair radiation damage to a certain degree; however, some damage cannot be repaired and accumulates in the tissues. In other words, radiation itself does not accumulate, but some of the un-repaired damage might. This can lead to future health problems such as the development of cancer, cataracts, birth defects, or premature aging.

  At one time it was believed that very low doses of radiation were not harmful. It was thought that there was a certain threshold, below which, no biological damage occurred. However, it now appears that there is no safe level of radiation. The low doses received by the patient from dental radiography produces very little damage, but it does occur. The number of cells in the body that are affected is low and the probability of cell death is even lower. Nevertheless, it is necessary to keep exposure to X-ray radiation to a minimum.

  The amounts of radiation involved in dental radiography are very small, much smaller then one *gray* or *sievert*. Just as a meter can be divided into centimeters or millimeters, the units of radiation measurement can be similarly divided. For example, a centigray is one hundredth of a gray.
- **Long-term:** Effects of radiation depend on the amount of damage caused to the vasculature. Small blood vessels undergo swelling, degeneration and necrosis. This initiates slow fibrosis and obliteration of the lumen of the **blood vessel**. These in turn results in low nutrition at cellular level causing low resistance and proneness to infection and trauma.
- **Amounts of radiation used in dental radiography:** The amount of radiation exposure produced when taking dental radiographs varies. It is dependent on film speed, technique, kilovoltage used, and whether any additional collimation is present. Speed "E" film requires only about one-third to one-half as much radiation for a diagnostic image as does speed "D" film. Higher kilovoltages and longer source-to-film distances (SFD) result in lower skin doses than do lower kilovoltages and shorter distances. Rectangular collimation further reduces the **amount** of tissue exposed.

  Techniques that employ intensifying screens generally require less radiation. The exposures in panoramic radiography vary from site to site, but they are often not higher than 10 or 15 milligray. In fact, for most areas of the head and neck, the dose is less than 5 mGy. The absorbed skin doses associated with skull films are only about 0.5 mGy. Absorbed doses to the marrow and other deeper structures are lower than the skin doses with all techniques.

SOURCES OF RADIATION

We are exposed to radiation everyday of our lives. Background radiation comes from natural sources such as radioactive materials in the ground and cosmic radiation from space. It also comes from man-made sources such as radioactive waste and nuclear fallout. The average background radiation is approximately 1.0 to 1.2 millisievert (mSv) per year; it varies slightly with geographic location. Areas of higher elevation receive more cosmic radiation, like people living in Nepal and the Himalayan belt in North and Northwestern India. The background exposure does not include the radiation from diagnosis or treatment (Table 24.1).

The natural sources of radiation include the sun and the stars and are termed as the cosmic sources of radiation. Mother earth has a lode of radioactive material like one seen in the coast of south India. The beach sands of Karunagappalli taluk in Kollam district in Kerala, Colachel taluk of Kanyakumari district in Tamil Nadu and Gopalpur in Ganjam district of Orissa has among the richest monazite deposits in the world.[7]

The radioactive black sand in Kerala, southern India where thousands of traditional fishing families have lived

Table 24.1: Sources of radiation

Natural	3 mSv		Artificial	0.60 mSv	
External		Internal	Medical	Consumer products	Others
Cosmic	Terrestrial	Radon			
0.27 mSv	0.28 mSv	2.0 mSv	0.55 mSv	0.10 mSv	0.04 mSv

The doses received from medical diagnostic radiology is one sixth that of background radiation, and hence may be told to the patient as safe

Taken from NCRP reports and other sources in India
These are approximate and may vary from geographical region
To geographical region in different parts of India

for generations are being exposed to the highest levels of natural radiation in the world. The glistening black sand on the beaches contains radioactive materials such as thorium and monazite. There are similar natural radiation areas in southern China, Iran and Brazil, but the Kerala coast in southern India is believed to be the only high radioactivity region with a high population density.[8]

RISK VERSUS BENEFIT OF DENTAL FILMS

Critical Organs

As we have seen, the radiation doses from dental radiography are small. However, there is a potential for biological damage every time tissues are exposed to radiation. There are some tissues or organs that are exposed to more radiation than others when dental films are taken. These tissues or organs are termed as the **critical organs.**

- The **skin** is the first tissue exposed to radiation from sources outside the body. One of its responses to radiation exposure is reddening or erythema. This is the same reaction seen in sunburn. Exposure to radiation increases the risk of skin cancer. Increased risks for skin cancer have not been demonstrated for doses of X-radiation less than 250 mGy. The dose to the skin of the face is about 10mGy when taking dental films using an open-ended cylinder and speed "D" film. Therefore, a patient would have to receive 25 complete mouth radiographic series (CMRS) in a very short time to significantly increase the risk of skin cancer.
- Radiation to the **lens of the eye** may produce cataracts (a cloudiness of the lens). The X-ray dose associated with this problem appears to be about 2 Gy (2000 mGy). The dose to the eye from a CMRS, using an open-ended cylinder and speed "D" film, is only about 0.6 mGy. Many scientists no longer consider the lens a critical organ in danger.
- The **thyroid gland** is fairly resistant to radiation in the adult. However, thyroid cancer has been found in people who were exposed to a dose as low as 0.05 Gy (50 mGy) when they were children. The dose to the thyroid from a CMRS (open-ended cylinder, speed "D" film) is only about 0.25 mGy. This dose to the thyroid can be further reduced by about half with the use of a thyroid collar. The use of a thyroid collar should be mandatory for children, since their thyroid tissues are more radiosensitive.
- Malignant changes in **bone marrow** may result in leukemia. There is active (blood-cell-producing) marrow in the mandible, skull, and cervical spine. About 13 percent of the total bone marrow lies in the head and neck areas. The dose to the bone marrow in a full-mouth series of radiographs (open-ended cylinder speed "D" film) is about 0.15 mGy. The X-ray dose associated with leukemia is about 50 mGy.
- The genetic effects of radiation can have far reaching results. However, the dose to the **reproductive cells** from dental radiography is very small, only about 0.005 mGy or less for males and 0.003 mGy for females. The female dose is lower because the reproductive cells are in more protected body location. If the patient wears a lead apron, exposure to the reproductive cells is virtually zero (0.000-0.0003 mGy).
- The exact amount of X-radiation that may produce damage to a developing human embryo is unknown.

However, doses below 100 mGy produce very little risk. Nevertheless, it is advisable to postpone non-emergency radiographs until after pregnancy. A minimal number of films for urgent care can be taken if the patient wears a lead apron.

- In summary, there is some risk attached to the use of ionizing radiation on biological tissues. However, the levels of radiation involved in dental radiography are only about 1/25 to 1/1,000 of the levels associated with injury. Therefore, the benefit of detecting disease in a patient, disease that might not otherwise be detected, far outweighs the risks of receiving small doses of radiation if the radiographs prescribed are exposed and processes in an appropriate manner.

RADIATION PROTECTION FOR THE PATIENT

Despite the low risks to the patient from dental radiography, it is best to keep exposure to ionizing radiation to a minimum. Therefore, the **ALARA** concept should be kept in mind when exposing dental films. This can be done by:

- **Film selection:** Fast film should be used. Only the type 'E' or the Ektaspeed is recommended in dental practice today, since it reduces the exposure by at least 40% as compared to type D.
- **Intensifying screens:** For extra oral films, lateral cephalogram, OPG, lateral oblique, etc. use of rare earth screens has reduced the dosage definitively.
- **Grids:** The use of grids reduces the fogginess of the film due to secondary radiation, thereby reducing the need for repeat films.
- **X-ray machines:** Only the units, which are manufactured by reputed companies, should be used.
- **Kilovoltage:** Using an X-ray beam with low kilovoltage results in higher patient doses, primarily to the skin. They do not reach the film and therefore do not contribute to the diagnostic image. Units should be operated using at least 60 to 90 kVp.
- **Filtration:** Units operating at 70 kVp or above should have filtration equivalent to 2.5 mm of aluminum. Units operating below 70 kVp should have the equivalent of 1.5 mm of aluminum. Filtration removes the low energy X-rays from the beam. These "soft" X-rays are absorbed by the patient and do not contribute to the image; removing them before they reach the patient, reduces radiation exposure.
- **X-ray collimation:** The beam should be collimated so that it is no more than 7 cm (2.75 inches) in diameter at the patient's face. Rectangular collimation further reduces the amount of tissue irradiated.
- **Position-indicating devices:** Open-ended, circular or rectangular leadlined cylinders are preferred for directing the X-ray beam. A long (12 to 16 inches) position-indicating device (PID) will reduce exposure to the patient better than a short (8 inch) PID, because there will be less divergence of the beam. Pointed plastic cones are not recommended.

The X-rays interact with the plastic and increase the amount of scatter radiation.

- **Film-holding devices** are recommended. They usually result in a more stable positioning of the film. In addition, the patient's hands are not exposed to radiation. If rectangular collimation is being used, a film holder and positioning guide is necessary.

Retakes should be kept to a minimum. If you are in doubt about the placement of a film or the position of the tube head don't press that button. Be sure you are taking the best film that you can.

- **Proper processing:** Use of quality solutions, well-designed dark room and application of the knowledge of X-ray processing technique with a healthy dose of common sense will optimize the processing.
- **Proper indexing and storage of the films:** In a large radiology department films could be lost due to carelessness, mismanagement or improper filing. Now in most institutions and even clinics, the storage of images in computers obviates all these medieval problems.
- **Miscellaneous:** Lead aprons should be used on all patients. Thyroid collars should be used on patients when intraoral films are being taken. Sound professional judgment can help minimize patient exposures.
- **RVG**: Recently the use of radiovisiography or RVG has further reduced the dose of the radiation required in the IOPA with the CCD sensors. The image appears directly on the computer screen and can be saved as a picture file on the hard disk. Printout on a regular paper is possible. For details see the chapter on digital radiology/computers in dentistry.

OPERATOR PROTECTION FROM RADIATION

People who work with radiation (that includes you) are also entitled to protection from radiation. There are exposure limits for occupationally exposed radiation workers. The ***Maximum Permissible Dose*** **(MPD)** is the dose of radiation to the whole body that produces very little chance of somatic or genetic injury. The MPD for whole body exposure per year for occupationally exposed personnel is 0.05 Sv (5 rem), An age-based formula has also been developed as guideline for any accumulated dose (N in years).

$$MPD = (N—18) \times 0.05\ Sv/yr$$

Occupationally exposed women who are pregnant are allowed an MPD of only 0.005 Sv/yr. This is the same dose limit that applies to the general population. Therefore, during their pregnancy, female radiation workers are treated like the general population and should receive far less radiation.

- **Planning and designing** of a safe Maxillofacial Radiology Department:
  - Radiation area should be at one corner in the building such that at least two walls open to the environment.
  - One extra thickness of brick with Barium plaster is a must for the walls.
  - Warning board and light should be seen, when the machines are operating, at the entry.
  - The barriers should have 2 mm or more of lead and it should go at least 12 inches below the ground (Fig. 24.1).
  - all the timers, control consoles should be kept behind the lead barriers.
- **Conch shell design:** The operatory that contains the X-ray unit should be constructed in such a manner that it protects people in surrounding areas from radiation. We recommend a conch shell design see figure below (Fig. 24.2).
- **Education and orientation** of all the radiation personnel to the ill effects of radiation is mandatory. Continuing education programs for the technicians will keep them alert to the possible risks and orient them to new equipment.

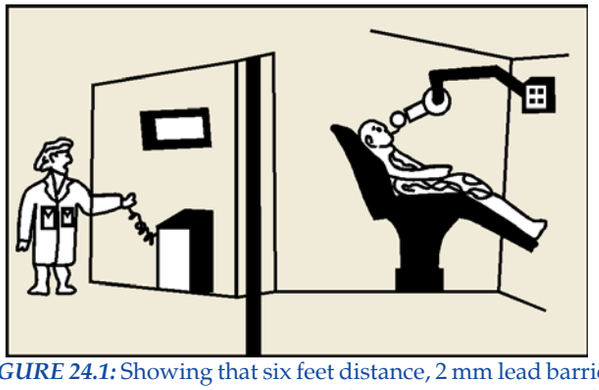

FIGURE 24.1: Showing that six feet distance, 2 mm lead barrier and lead incorporated at least one feet below the ground for secondary radiation protection (Prasanna K, Bailoor DN 2004, Yenepoya Dental College and Hospital, Mangalore

- **Film badge service** is a good way to keep track of occupational exposure. Badges are worn by personnel at all times while at work, and are regularly sent to the company providing the service. Written reports of the exposure recorded on the badges are provided. If proper safety precautions are followed, no one in a dental office should receive radiation doses close to their MPD.

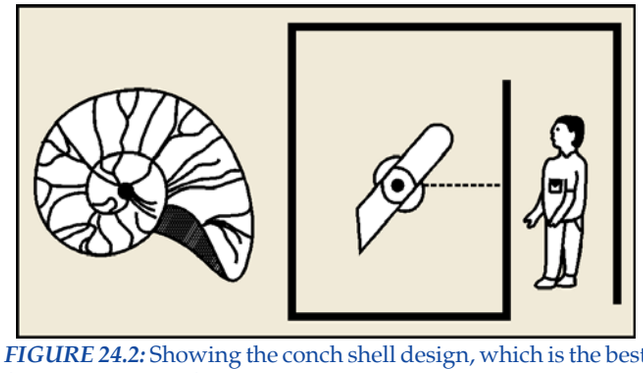

FIGURE 24.2: Showing the conch shell design, which is the best for the planning of the radiation, protected areas in one corner of the hospital, such that at least two walls are outside (Prasanna K, Bailoor DN 2004 Yenepoya Dental College and Hospital, Mangalore)

- **Lead barrier**: It is preferable that the operator stands behind lead barrier while exposing films. The barrier should have a window or other means of monitoring the patient during the exposure. If no barrier is available, the operator should stand at least 6 feet away from the patient and in an area that lies between 90 and 135 degrees to the primary beam. These are areas of minimum scatter radiation (Fig. 24.3).

- **Never hold the film or tube:** Dental personnel should never hold films for patients. If assistance is necessary, ask a family member or guardian to help. Be sure to protect the helper with lead apron as well. Dental personnel should also never hold the tube head for stability. If the equipment is that "wobbly", it should not be used until professionally repaired.

Most dental offices have a fairly low radiation workload. This means that low milliamperage and exposure times are used. Shielding or barrier requirements are based on workload, kilovoltages used, distances involved. Barium plaster used in the construction stage during the brickwork reduces the radiation in adjacent areas. Wood paneling or veneering alone does not provide adequate protection. Dental surgeons are referred to the NCRPM (National Council on Radiation Protection and Measurements) publication no. 35 for specific details.

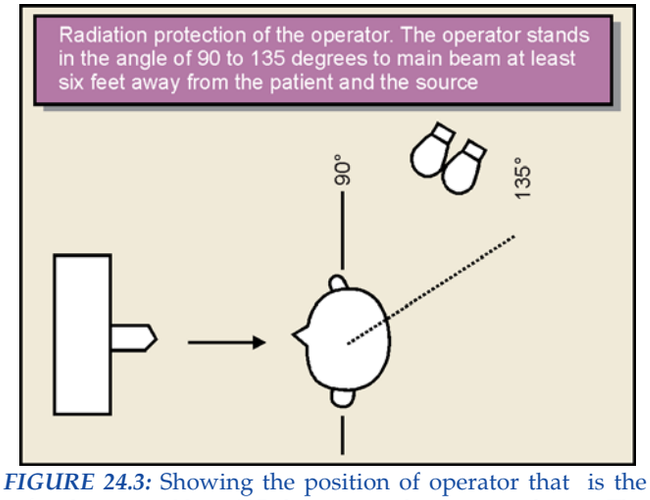

FIGURE 24.3: Showing the position of operator that is the safest between 90 to 135 degrees to the primary beam. The distance of six feet and at least 2 mm lead barrier in addition can cut down the radiation to ALARA levels (Omal PM , Bailoor DN 2004, Yenepoya Dental College and Hospital, Mangalore)

EFFECT OF THERAPEUTIC RADIATION

Oral cancer is treated by radiotherapy in regions of nasopharynx, floor of the mouth, soft palate, etc. This is used when the tumor is radiosensitive, deeply invasive, and due to anatomical peculiarities surgically difficult to approach. Most of the oncology centers now use combined surgery, and chemotherapy together with radiotherapy for optimal results.

Normally the dose to the oral cancer is given in small increments called as *Fractions.*

Fractionation allows the cells to undergo repair in the normal tissue region.

This increases the O_2 tension in the tumor cells making it more radiosensitive. Ellis[1] has introduced us to the concept of NSD or the ***Nominal Standard Dose***-NSD is the single dose that produces a similar clinical skin reaction to that produced by a given fractionation scheme. The unit of the NSD is ***ret*** (rads equivalent therapy).

Effects of therapeutic radiation on oral tissues; (for oral cancer) the dose given are 200 R per day upto 5000-6000 R delivered through 8 × 10 cm fields. Here in Mangalore, Bangalore and Bombay, cobalt units are used. However, on occasions Radon or Iodine 125 can be placed directly into the tumor mass. The therapeutic radiation given from a distance by a cobalt unit is termed as teletherapy and when the radioactive materials are inserted directly into the tumor mass it is termed as brachytherapy.

- **Oral mucous membrane (OMM):** Four to fifteen days after undergoing therapeutic radiation, mucositis sets in.

  By the third week most of the patients show a yellow pseudomembranous slough. Good oral hygiene minimizes the infection, topical anesthesia is required at meal times, antifungal Rx and artificial saliva may help in combating the resultant xerostomia. Mucosa heals two months after the radiation therapy but still remains pale and atrophic. This probably results from the slow fibrosis and atrophy of the vasculature in the submucosa regions.
- **Taste buds:** These are sensitive to therapy resulting in loss of taste acuity. The dysgeusia becomes more severe due to xerostomia and change in viscosity of saliva.

  100 to 120 days after the stoppage of radiation the taste returns to normal.
- **Salivary glands:** Parenchymal part of the gland is radiosensitive and exposure of 25 to 30 Gy results in an acute inflammatory response after starting the therapy. When 60 to 70 Gy of radiation is continued chronic inflammatory response sets in, resulting in loss of fine vasculature and in parenchymal degeneration.

Saliva shows elevated Na^+, Ca^{++} Mg^{++} and pH of saliva averages 5.5 compared to 6.5 in normal patients. This increases the acidogenic bacteria like streptococcus mutans and lactobacillus.

- **Teeth:** Adult teeth are resistance to effect of radiation exposure. In acute radiation the pulp may undergo necrosis and fibro-atrophic change. During development, if radiation precedes calcification, severe malformation, retarded root development and destruction of entire tooth buds may result. A dose of 200 R can cause hypoplastic enamel. Radiation caries is rampant form of dental decay, which is associated with those individuals who receive radiation in the facial region.

Three types of caries are observed:

- Involving cementum and dentin in the cervical region.
- Generalized superficial caries affecting the buccal, Occlusal, incisal and palatal areas of the teeth.
- Dark pigmentation of the entire crown and increased Occlusal and incisal wear.

In clinical practice we see combination of these three types in various regions.

In countries with better health awareness like USA, UK, etc. only grossly carious teeth and periodontally involved teeth are extracted and the rest saved by a combination of restorations, oral hygiene and use of fluoridated viscus gel and hexidine mouth washes. In India it is observed that many of the patients of oral cancer who take radiation come from poor socioeconomic class with severe tobacco and alcohol abuse combined with nutritional compromise. In such cases more often than not, doing a total dental extraction is a gentle alternative to treating the osteoradionecrosis at a later date.

- **Bone**: The radiation damages the fine vasculature of the already poorly supplied dense bone of the mandible, causing it to become hypoxic, and the normal marrow is replaced with the fibrous tissue. The decreased vascularity results in the necrosis due to decreased vitality of the bone and the increased proneness to infection. This bone infection after the radiation process is termed as osteoradionecrosis (ORN).

Factors that may precipitate ORN are:

- Higher the radiation dose
- Post-irradiation extractions
- Non-evaluation of the existing periodontal disease
- Lack of proper oral hygiene by patient due to ignorance or carelessness
- Presence of complicating metabolic factors like diabetes mellitus.

All extractions and alveolectomies must be attempted by a dental surgeon at least 15 days prior to the start of the radiation therapy.

In patients whose mandible might have received more than 65 Gy of radiation must not undergo extractions, but endodontics is superior alternative, in fact this situation normally demonstrates that the dental surgeon was not consulted prior to the radiation planning.

Treatment of osteoradionecrosis is done by use of hyperbaric oxygen chambers, Davis J et al (1979)[2] used antibiotics like Clindamycin and Vancomycin for 7-10 days or more in a hospitalized set up, Myocutaneous flaps containing fifth ribs have been used with some success by Pearlman N et al (1983)[3] In addition powerful analgesics like the Fortwin®,or Pentawin® need to be given for this extremely painful condition.

WHOLE BODY RADIATION

When the whole body is exposed to radiation in wars, nuclear accidents, or astronauts in outer space during solar storms, a very characteristic change takes place in the human body. These changes have been noticed in the other mammals also. This has been called as Acute radiation syndrome.[5]

Acute radiation syndrome is divided into:

- Prodromal syndrome .....0.5-1.0 Gy
- Bone marrow syndrome .....2.00-10.00 Gy
- Gastrointestinal syndrome .....10-100 Gy
- CNS syndrome .....Excess of 100 Gy
- **Prodromal syndrome**: Generalized GI tract problems like nausea, vomiting, diarrhea, anorexia normally seen within one gray of full body exposure.
- **Bone marrow syndrome:** The range of radiation, around 2-10 Gy injures the bone marrow. This depresses the circulating cells of all populations. Lymphopenia, granulocytopenia, and anemia may be clinically evident. Death may occur 10-30 days after radiation.

Following the moderate injury 7-10 days before clinically significant leukopenia develops. The dental surgeon can and should remove all the sources of infection (e.g. pericoronal flaps, periodontal abscess, or grossly carious teeth) and should administer bactericidal antibiotics like Megapen, or Bipen, Ampicillin+Cloxacillin combinations. This, combined with transplantation of bone marrow, may save some individuals from acute radiation syndrome.

- **Gastrointestinal syndrome**: The range of 10-100 Gy damages the GI tract. The similar prodromal syndromes are accentuated namely anorexia, nausea, vomiting, severe diarrhea, dehydration and prostration. Ulcers may perforate in the GI tract resulting in septicemia and acute abdomen, before turning fatal.
- **Central nervous syndrome (CNS syndrome)**: Exposure more than 100 Gy causes death within 24-48 hours. Stupor, convulsions, disorientation and motor in coordination; this is the result of the direct damage to neurons and indirect effect of radiation necrosis of the vasculature supplying the brain. The patient normally dies before any treatment can be given and before either GI tract syndrome, or bone marrow syndrome can manifest.

The concept of latent period and doubling dose:

- *Latent period:* It is the time elapsed between the exposure to any dose of radiation and discernible clinical manifestations that may be attributed to it.
- *Doubling dose:* It is the amount of radiation that will stimulate, as many mutations additionally as occurring spontaneously, in that population.

Human genetic doubling dose has been calculated as

Males = 0.46 Sv

Female = 1.25 Sv

SUMMARY

The effects of radiation on man are divided into **Genetic** and **Somatic** effects. The diagnostic dental radiation is determined to be safe by many studies for the patients, but the additive effect on the dental surgeons and dental technicians is still in the area of speculations, certain concepts need to be assimilated at this juncture.

An average person receives low radiation from diagnostic and background radiation, so these two groups do not seem to contribute to the genetic damage in human populations.

Long-term effect of the low-grade ionizing radiation on the body of human population has proved difficult to study due to very subtle changes and long latent period.

Cataract formation and life span shortening are two somatic effects that are at present under investigation. But safety of diagnostic dental radiography has been established in this area.

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25 Radiographic Investigation

INTRODUCTION

There are three main types of intraoral radiographs:

- Intraoral periapical radiograph
- Bitewing radiograph
- Occlusal radiograph.

The anatomic area of interest and the type of pathology suspected helps the clinician to decide the type of radiograph to be taken.

The *intraoral periapical radiography* (IOPA) is the basic investigation that gives graphic information about the alveolar bone, periodontal areas and the hard tissues of the tooth. The mastering of the technique, for making good IOPA radiographs is essential for the dental surgeon, both for routine and specialist work.

Two intraoral projection techniques may be used for periapical radiography:

- *Paralleling cone* and
- *Bisecting* angle

Paralleling cone technique: It is also called long cone technique or right angle technique. The rationale is the central ray of X-ray beam is directed at right angles to the teeth and the film. The X-ray film is kept parallel to the long axis of the teeth. So special holders which keep the film parallel to the long axis of the tooth are utilized. A long cone of 12 inches is used. The kVp used is usually 85-90 kVp. The X-rays are directed perpendicular to the film and therefore there is minimum geometric distortion, less magnification and more definition (Fig. 25.1).

The film holding devices used in paralleling cone technique could be:

The XCP® instruments (extension cone paralleling) The precision rectangular collimating instruments which restrict the beam size at the patient's face to size of the radiography. The stab disposable film holder. The Snap-A-Ray® intraoral film holder A hemostat inserted through a flattened rubber bite block which will serve in much the same manner as the Snap-A-Ray® film holder.

Brocklebank LM (1998)[2] has outlined beautifully the various factors that go into formation of a good dental radiographic image. The practitioner is directed to read this update.

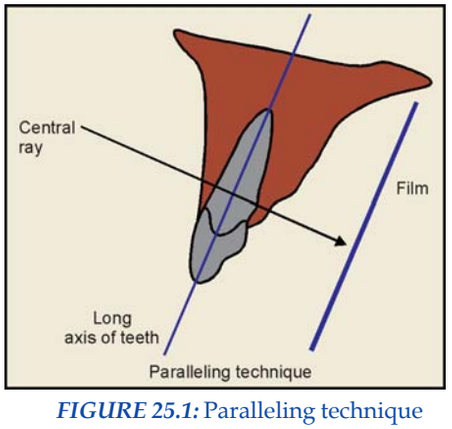


FIGURE 25.1: Paralleling technique

Bisecting Angle Technique

Involves taking radiographs by keeping the head in anatomically fixed position, and such that the long axis of the rays are perpendicular to the bisector between the long axis of the tooth and long axis of the film (see Fig. 25.2). An eight inch cone is normally used. In India we prefer to use open ended cones with Ekta speed 'E' films. kVp used is usually 55-65 kVp.

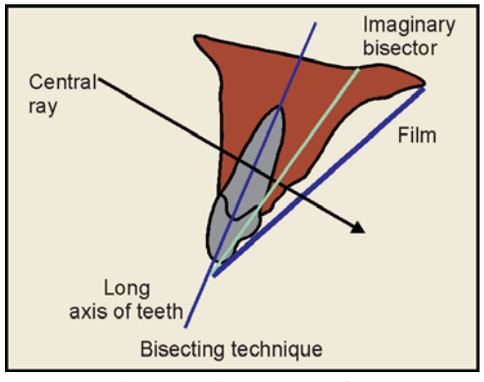


FIGURE 25.2: Bisecting technique

The bisecting angle technique is based an a simple geometric theorem; Cieszynski's rule of isometry, which states that two triangles are equal when they share one complete side and have two equal angles (In addition, their corresponding sides are equal). In dental radiography the theorem is applied as follows: the film is positioned as close as possible to the lingual surface of the teeth, resting in the palate or in the floor of the mouth. The plane of the film and the long axis of the teeth form an angle with its apex at the point where the film is in contact with the teeth. When the angle is bisected by an imaginary line or plane, two congruent angles, with a common side (the imaginary bisector), are formed. A line, representing the central ray of the X-ray beam) will complete the third side of two triangles when it is directed (through the apices of the teeth) perpendicular to the bisecting line (Fig. 25.2).

Angulation of Tube Head

The position of the X-ray machine tube head is usually adjusted in two planes in a vertical and a horizontal movement.

In the paralleling technique the guide planes of the instruments are used, which gives the clinician an accurate place to keep the open ended cone. This is very convenient with the physiologic type of dental chairs, since bisecting angle technique usually requires the patient to be in upright position.

Horizontal position of the tube head is zero degrees by convention and cone pointing downward is positive and upward is negative. Logically all the maxillary radiographs will require positive angulation and mandibular radiographs negative or zero.

General Steps for Making an Exposure (Bisecting Angle Technique)

The films used here is Adult = 32 × 41 mm, Children = 2 2 × 35 mm.

Courteously Seat the Patient

The patient is seated comfortably such that the arch to be radiographed is parallel to the ground. The mid-sagittal plane should also be perpendicular to the ground.

The dental chair should be lowered for maxillary projections and elevated for mandibular projections. The patient should be asked to take out all the removable appliances from their mouth and spectacles if any.

The patient should be draped with a lead apron or with a U shaped lead protective trade name "Protectoray®" even for a single IOPA. The supine position is occasionally used in intraoral radiography instead of having the patient sit upright. This may most easily be accomplished by using reclinable dental chairs. The use of the supine position does not affect the frequency and distribution of technique errors compared with using paralleling instruments in the upright position. There is, however, marked reduction in the patient's reactions to the supine position have been reported as generally favorable, especially for apprehensive patients.

- **Barrier method for cross infection control**: Cover the dental chair, unit and the X-ray cone head with disposable plastic wrappers, which are pre-sterilized. The patient is covered with a disposable long Bib.
- **Adjust the X-ray unit setting:** The X-ray machine should be set for the proper kVp, mA, and exposure time according to the recommendations of the film manufacturer or that which experience has demonstrated

to produce the highest quality film with the least radiation.

- **Position of the tube head:** Bring the tube head to the side to be examined so as to have it readily available after the film has been positioned.
- **Wash hand thoroughly:** Wash your hands with soap and water, preferably in front of the patient or at least in an area where the patient can observe or be aware of the washing and then wear gloves.
- **Examine oral cavity:** Before the film is placed in the mouth, examine the teeth to estimate their axial inclination. This will in turn influence the placement of the film. Also note tori or other obstructions that will modify film placement. In sensitive patients use local anesthetic cream in the area which will be adjacent to the film held.
- **Position of the film:** Remove the film from the film dispenser, insert it in the film holding device, and position the film in the region of the patient's mouth to be examined. The film should be routed into the oral cavity, leading with the apical end of the film. For the mandibular projections, especially of the anterior teeth, will usually displace the tongue by mentioning it. The film in the holding instrument should be gently lowered into the mouth until it just touches the floor of the mouth or palatal area.

  Do not try to displace the floor of the mouth with the film at this time. With the film resting gently on the floor of the mouth rotate the instrument until the bite block meets the lower teeth. At this point, in positioning the film, it will usually be at a large (unsatisfactory) angle with the long axis of the teeth, and the extraoral portion of the film holding instrument will be conspicuously tipped inferiorly. However, if the patient is asked to close the mouth slowly, and as he or she closing, the instrument can be slowly rotated superiorly, the film will be lowered into the floor of the mouth so that when the instrument is stabilized by the teeth the film will be deep in the relaxed floor and much closer to parallel with the teeth than would have been possible without this maneuver. When positioning film for mandibular projections, an effort should be made not be touch the extremely sensitive attached gingiva on the lingual aspect of the alveolar process. Horizontal angulation is measured from the transcondylar line to the X-ray beam as seen from above.

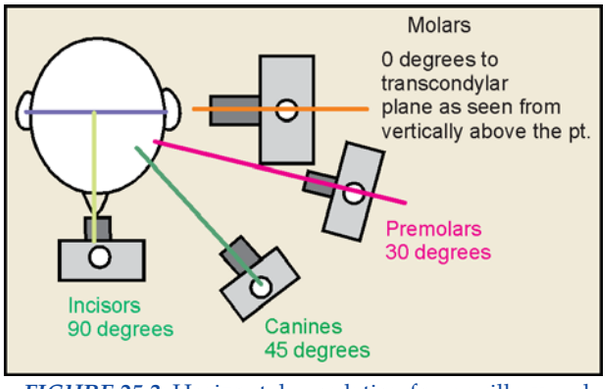


FIGURE 25.3: Horizontal angulation for maxillary and mandibular teeth IOPA radiography

- **Position of the X-ray tube:** Adjust the vertical and horizontal angulation of the tube head to the beam-guiding instrument. When a beam-guiding instrument is not used, aim the central ray at the appropriate entry point on the skin (identified in the figure). It is wise to caution the patient not to move (Figs 25.3 to 25.7).

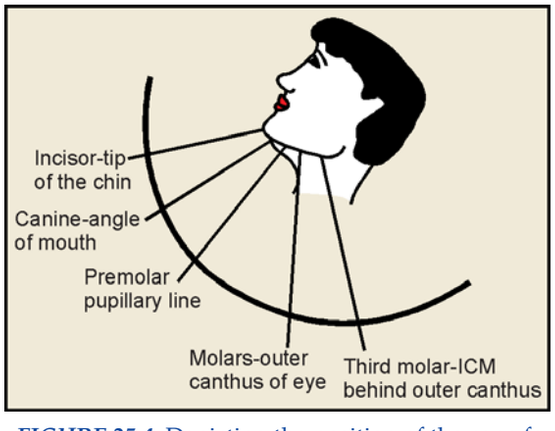


FIGURE 25.4: Depicting the position of the cone for mandibular IOPA radiography

- **Make the exposure:** After exposure, remove the film from the patient's mouth, dry it with a paper towel, and place it in an appropriate receptacle protected by a lead lining.
- **Remove the plastic barrier** material by ripping it away and putting it into the proper receptacle for incineration. Ask the assistant to prepare the chair and tube for the next patient.

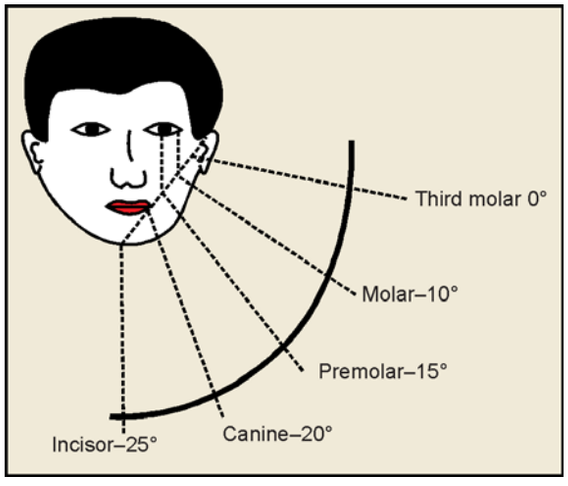


FIGURE 25.5: Recommended vertical angulation in bisecting technique

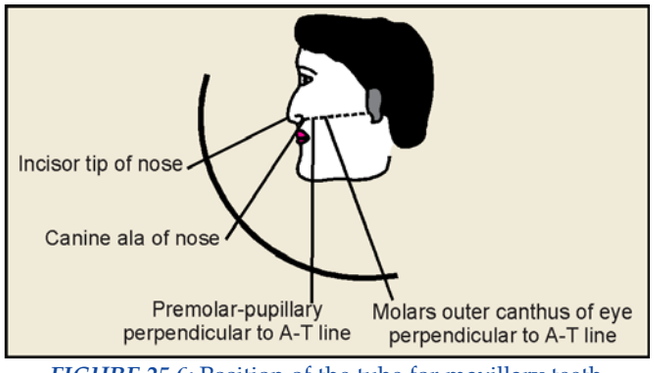


FIGURE 25.6: Position of the tube for maxillary teeth

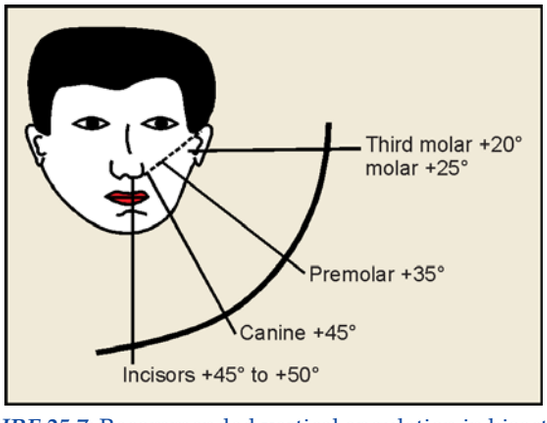


FIGURE 25.7: Recommended vertical angulation in bisecting technique for maxillary radiographs

BITEWING EXAMINATION

The bisecting angle technique requires the primary ray to be at an angle which varies from area to area, and which is non-perpendicular to the long axis of the tooth. This does not allow good visualization of initial lesions of inter-proximal caries, as well as periodontal lesions. Bitewing Radiography is that intraoral technique which allows the clinicians to evaluate initial lesions by passing the primary ray perpendicular to the long axis of the respective teeth (Figs 25.8 and 25.9).

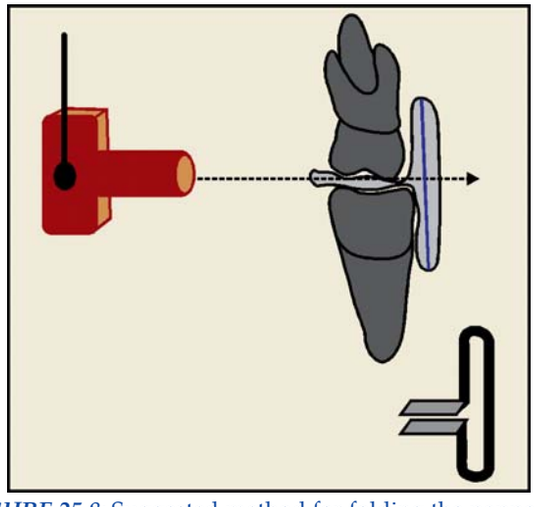


FIGURE 25.8: Suggested method for folding the papper tab

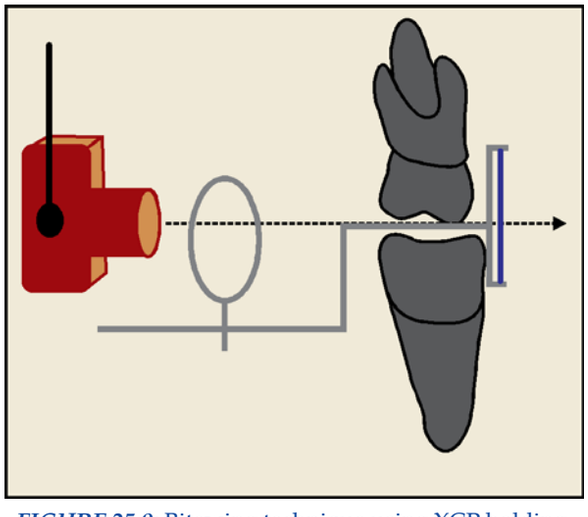


FIGURE 25.9: Bitewing technique using XCP holding instrument

Indications for Bitewing Technique

- To screen for incipient proximal caries
- To check the health of the inter-dental alveolar bone in normal and periodontal disease
- To detect calculus deposits in inter-dental areas

FIGURES 25.10A and B: Showing XCP holding instruments and technique (Sonali Bailoor, Bailoor DN 2004, Yenepoya Dental College and Hospital, Mangalore)

- Detection of secondary caries under the restorations
- To determine if restoration is fractured
- Relationship of deciduous to the permanent in children with mixed dentition
- Routine annual evaluation of all patients who come to check up without any complaint.

The standard adult size periapical film can be fitted with a bitewing tab and used for this examination. There are also film-holding instruments that support the film and provide an external reference for positioning the tube head (Fig. 25.10).

OCCLUSAL RADIOGRAPHY

- In general the indication for an occlusal radiograph is a requirement to visualize a relatively large segment of a dental arch, including the palate or floor of the mouth and a reasonable extent of the contiguous lateral structures (Fig. 25.13).
- This technique involves the superoinferior visualization or *vice versa* on a radiograph which is 2½'' × 3'' (57 mm × 76 mm). Which means that it is possible to see the buccolingual or buccopalatal relationship accurately.
- To precisely located roots, supernumerary, un-erupted, and impacted teeth. This technique is especially useful in the cases of impacted canines and third molars.
- To localize foreign bodies in the jaws and stones in the ducts of salivary glands.
- To demonstrate and evaluate the integrity of the anterior, medial and lateral outlines of the maxillary sinus.
- To aid in examining patients with trismus who can open their mouth only a few millimeters, thereby precluding the use of the intraoral radiography.
- In providing information relative to the location, nature, extent, and displacement of fractures of the mandible and maxilla.
- To determine the medial and lateral extent of pathoses (e.g. cysts, osteomyelitis) and to detect their presence in the palate.

The intraoral occlusal radiograph is made by inserting a relatively large film between the occlusal surfaces of the teeth, in the plane of occlusion (hence the name occlusal radiograph). The "tube" side of this film should be turned

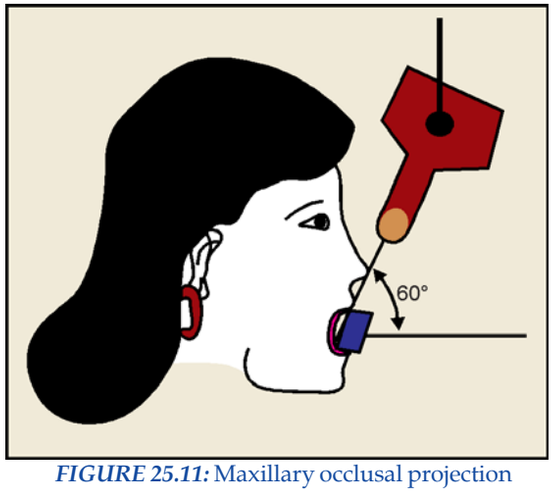


FIGURE 25.11: Maxillary occlusal projection

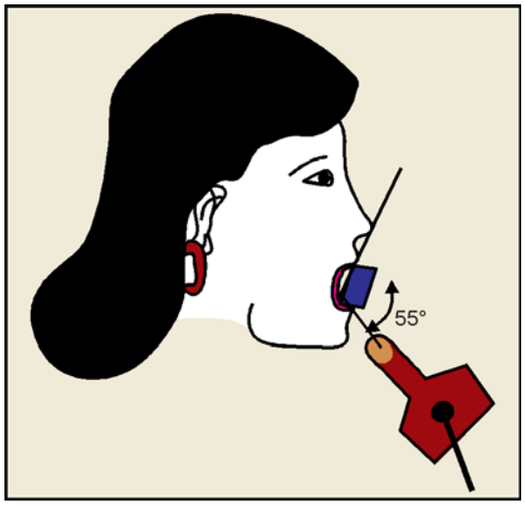
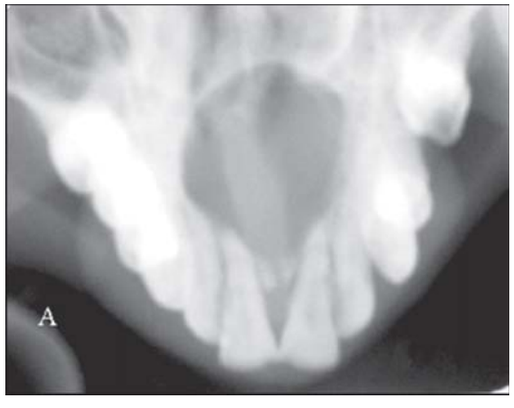
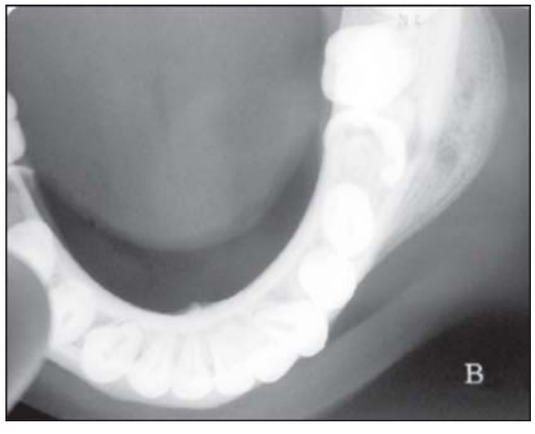


FIGURE 25.12: Mandibular occlusal projection





FIGURES 25.13A and B: A. Shows the occlusal view of the maxilla showing a large radiolucent lesion most probably a incisive canal cyst. B. Mandibular occlusal showing bony hard swelling of the left side with onion peel appearance associated with Garres osteomyelitis (Kartikeya Patil , Mahima Patil JSS Mysore 2004)

toward the jaw to be examined, and the X-ray beam directed through the jaw to the film. The size of the film will accommodate relatively large portions of the jaw being examined. In general, standardized projections are used whereby there is desired relation between the central ray, film, and region being examined. The clinician should feel free, however, to modify these relations as indicated by a specific clinical requirement (Figs 25.11 and 25.12).

EXTRAORAL RADIOGRAPHIC EXAMINATIONS

The common extraoral views of use to a dental surgeon are:

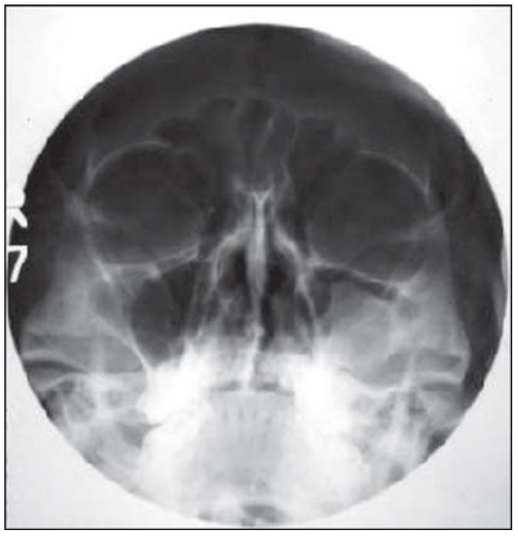
- Orthopantomogram
- Paranasal sinuses view
- PA mandible
- Lateral oblique view mandible
- Temporomandibular joint views
- Reverse towne projection
- Submentovertex projection- the jug handle view.

Projection

- *OPG*: The orthopantomographic view is good for over all appraisal of the jaws of the children and the adults and details are discussed in Chapter no 29.

FIGURE 25.14: Paranasal sinus view showing a radiopacity of the left maxillary sinus most probably a mucosal cyst (Ani John, Hemant Umarji 2004 GDC Mumbai)

- ***Paranasal sinuses view***: Following structures are seen clearly in a well taken PNS view. The maxillary sinuses, frontal and ethmoidal sinus, orbit, Frontozygomatic suture and coronoid (Figs 25.14 and 25.15A).
  Technique: Standard 10 x 12 inch cassette is used with screens and with grid. Cassette is perpendicular to sagittal plane of the head, the canthomeatal line or the radiological base line 37 degrees above the horizontal. It is preferable to have the patient's mouth in open position. The central ray is projected perpendicular to the film at the level of maxillary sinus, 90 kVp with 120 mA s usually gives good picture of density and contrast.



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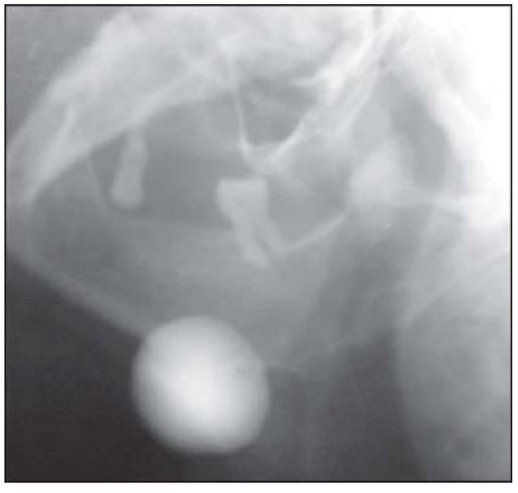


B

FIGURES 25.15A and B: A. Paranasal sinus view, B. Lateral oblique view

- **Lateral oblique projections:** An 5 × 7 inch cassette is used with the regular 60 kVp machine or regular dental X-ray machine. Two views can cover the mandible. Cassette is hand held by the patient or a special wooden platform can be made to place the angle of the mandible such that the ramus of the side radiographed is kept in close proximity to the film. Lateral oblique projection demonstrates the premolar-molar region and the inferior border of the body of the mandible. It provides much broader coverage than is available with periapical projections (Fig. 25.15B)
  Head position: The head is tilted toward the side to be examined, with the mandible protruded. The nose tip should be touching the cassette.
  Film placement: The cassette is placed against the patient's cheek and centered over the first molar, the lower border of the mandible and extend at least 2 cm below it. The cassette is held in place by the patient.
  Projection of central ray: The central ray is directed toward the first molar region of the mandibular body to be examined from a point just below the angle of the mandible on the tube side. The central ray should be perpendicular to the film.
  Exposure parameters: Although exposure parameters will vary, it is usual to use 65 kVp, 10mA, and about 0.90 second for par speed screens and medium-speed film.

  Sano K et al[3] 1998 have used a special occlusal X-ray film holder which could help to take a lateral oblique of the jaw. This apparatus can be easily made using indigenous materials and with customizable modification. Serious radiographers are referred to this text.
- **Posteroanterior view of mandible**: This provides good visualization of the mediolateral dimensions of the ascending ramus and the study of facial symmetry, frontal, ethmoidal, nasal fossa and orbits (Fig. 25.16).

  Head is positioned with radiological base line, i.e. the canthomeatal line parallel to floor. The cassette, 10″ × 12″ is placed in the forehead nose position, head holders are available to stabilize the head and the tube film distance preferred between 36 inches and 40 inches. The cone is such that central ray passes through the mid-sagittal plane of the head at level of the nasal

bridge. The kVp chosen is usually 75-80 and the exposure factors of 90 mAs normally are enough for regular screen films and with a grid 120 mAs normally are enough.

FIGURE 25.16: PA mandible

- **Reverse towne radiograph**: This view is used to see the subcondylar areas and, the posterolateral wall of the maxillary sinus. Head is placed such that the radiological base line is 25-30 degrees downwards. In our department we keep the patients mouth open using a plastic prop as much as comfortably can be opened. Central ray is directed perpendicular to the cassette in the sagittal plane, through the occipital bone. 75-80 kVp and 75-80 mAs is used with regular intensifying screen and about 90 mAs with grid is used. With rare Earth and compatible films 60 mAs also gives satisfactory results.
- **Submentovertex radiograph**: The SMV is useful for seeing the zygomatic arches, lateral wall of the antrum of highmore, the position and orientation of the pole of the condyle, sphenoidal sinus, medial and lateral pterygoid plates could also be visualized.

  Cassette with film is held in a positioner. The dental chair is oriented as far as possible and the neck of the patient is hyperextended backwards such that Frankfurt horizontal plane is parallel to the cassette. Midsagittal plane is kept perpendicular to the cassette and floor (Fig. 25.17).

  The central ray should pass through the floor of the mouth 1 inch from the chin and towards the vertex of the skull perpendicular to the film cassette combination. 75-80 kVp distance of 24 to 36 inches can be utilized and regular screen—film combination with grid requires mAs of 75-80.

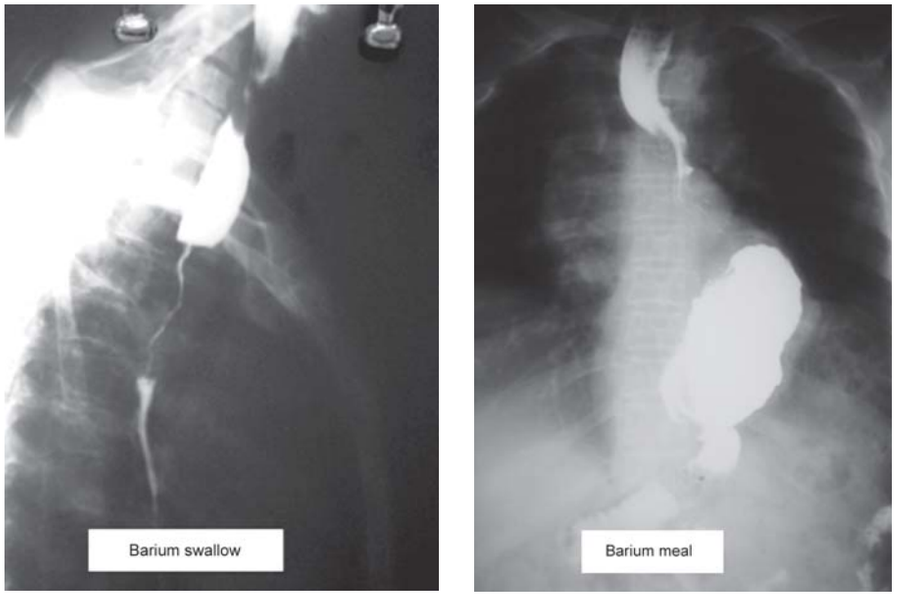
FIGURE 25.17: Showing the jug handle view. It is a SMV view taken with decreased tube film distance and lower kVp then the SMV. The zygomatic archs are highlighted and the fracture of the left arch is clearly evident in the above picture (Ani John, Umarji H GDC Mumbai 2004)

TMJ is visualized by using the following extraoral views.

- Transcranial
- Transpharyngeal
- Transorbital
- Corrected lateral tomography
- Panoramic radiography
- Arthography
- Magnetic resonance imaging (MRI)

SPECIALIZED IMAGING TECHNIQUES

Routine radiography of intraoral periapical variety, occlusal and orthopantomography has been superseded by the latest imaging techniques that are available at tertiary level hospitals.These are mainly contrast radiography, Magnetic resonance imaging (MRI), Radionuclide diagnosis (RND), Thermography, Ultrasound (US), Digital radiography, CT scan, endoscopic examination.



FIGURES 25.18A and B: Showing radiographic appearance of barium swallow and barium meal (Courtesy: Dr Ramachandran, RCC Trivandrum 2004)

Contrast radiography: The use of radiopaque contrast media for visualization of the soft tissue is refered to as contrast radiography. Following are the different types of contrast radiography.

- ***Cystography***: The radiographic visualization of soft tissue cysts after the injection of either Conray or Urograffin solution may be termed as cystography.
- ***Sialography***: The injection of radiopaque liquid material into the ductal system of the major salivary glands and its subsequent radiography is termed as sialography. It is indicated in the autoimmune sialoadenitis, salivary gland cysts and tumors.
- ***Arthrography***: The injection of the radiopaque dye into the superior compartment of the TMJ and its subsequent routine and tomographic examination is termed as the arthrography.
- ***Arteriography***: The localization of one of the major arteries and then injecting the radiopaque dye is termed as the arteriography.
- ***Barium swallow***: Making the patient swallow the barium solution and taking serial radiography is highly indicated in investigation of the dysphagia cases. Making a digital radiographic picture dynamically is termed as the cineradiography of the swallow process (Fig. 25.18A).
- ***Barium meal***: The visualization of large intestines in conditions like Peutz-Jeghers syndrome and Laugier Hunziker syndrome are important to establish the pathology like intestinal polyposis (Fig. 25.18B).

THERMOGRAPHY

It is the procedure by which the heat naturally emitted by the body is detected, measured and imaged. The resultant image 'thermogram' is a visualization of the distribution of the heat pattern of the body surface. This heat is naturally emitted as a result of either normal body function or disease or injury. The most commonly used method of thermography is the "infrared thermography", the heat (infrared) radiation emanating from the skin is collected in a manner similar to that used in light photography. The infrared is then electronically changed into a signal used to generate an image on a cathode ray tube (TV monitor). This image is then viewed in real time and the observations are used to properly adjust the equipment. The image is then recorded on a Polaroid or photographic film for viewing and interpretting the diagnostic findings. This method is called "Tele thermography".

- ***"Liquid crystal thermography"*** uses liquid crystal sensors to image the infrared picture.

- *"Computer assisted thermography"* uses computer program to determine the likelihood of disease.
- *"Microwave thermography"* measures and images microwave wavelength radiation emitted from the body.

Infrared radiation is an electromagnetic radiation emitted as a function of local temperature. Metabolism generates heat. Local metabolism is often increased in areas of malignant tumor; inflammation or injury, in order to obtain thermal equilibrium some areas of the skin will be warmer and other cooler. The temperature is represented in shades of gray, with warmer levels of temperature in lighter shades and cooler areas with darker area. But in most of the apparatus reverse of this can be achieved.

The image can be viewed as:

1. Lesions with normal temperature
2. Lesions with hypothermia- called as cold spot
3. Lesions with hyperthermia- termed as hot spot

Colored thermograms are being experimentally tried by many investigators, which are supposed to give higher resolutions. The color difference may represent color change as little as 0.1°C.

Gratt BM et al[6] have used this technique to differentiate the usual dental pain from Atypical odontalgia. Spread of infection in cellulitis and vascular headache, etc. has special imaging characteristics with hot spots in the affected region. This modality can complement the CT scan and MRI and give good insight for the differential diagnosis. An internal derangement of the TMJ is another area that requires the use of this modality. Prints can be taken on Polaroid film for records or observed on computer screen.

ULTRASOUND (US)

It is also called as ultrasonography which makes use of sound above the human hearing capability usually above 20,000 Hz. Thus it is distinguished from other mechanical waveform simply by having a vibratory frequency. For diagnostic purposes frequencies ranging between 1-20 MHz is used.

Transducers convert the electrical energy into ultra-high-frequency sound waves, these waves pass thorough the object and whenever they meet a surface of changed density they are reflected back partly, a part of this transducer also acts like a sensor and converts this returned energy into images.

The most important component of the transducer is a thin piezoelectric crystal or material made up of a great number of dipoles arranged in a geometric pattern. The most widely used piezoelectric material is lead zirconate titanate.

Sonic waves reflected back (Echoes) causes a changes in the thickness of the crystal, which in turn produces an electrical signal that is amplified, processed and displayed.

Techniques currently in use permit echoes to be processed at a sufficiently rapid rate to allow for the perception of motion. This is referred to as real time imaging. Ultrasound is good for superficial structures and when adjacent structures transmit sound at a different rate.

It is helpful in determining the outlines of structures but not necessarily their contents.

It is inexpensive, noninvasive, non-ionizing and produces no side effects.

Doppler ultrasound is particularly useful in identifying soft tissue vascular lesions.

Pavlov IP (2000)[7] reported that the accuracy of US in differentiating between benign and malignant lesions of the salivary gland is 91.8%.

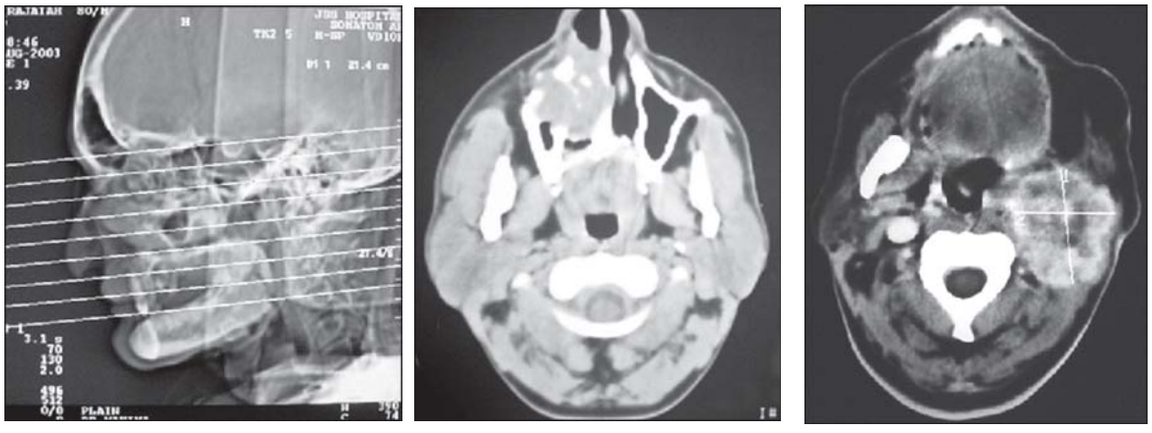
Koischwitz et al (2000)[8] has reported that US guided fine needle aspiration biopsy is a very accurate diagnostic procedure. It can be used for deep lymph nodes, salivary gland pathology and for deep seated hamartomatous lesions.

Autoimmune sialoadenitis was evaluated using US very efficiently by Salaffi et al (2000)[9].

The main application of US is the differentiation between solid and cystic masses. Solid masses are echogenic producing internal echos. Cystic or fluid filled lesions are echo free with enhancement of the deep wall. US is one modality, which is not being utilized to its fullest potential by the Oral Physicians and Oral Imageologists in the diagnosis.

CT SCAN

Godfrey Hounsfield was one first engineers to win a Nobel Prize in Medicine. Computerized axial transverse



FIGURES 25.19A to C: Showing CT image A. Scout image, B. Mass lesion of maxilla, C. Parotid lesion (Courtesy: Ramachandran RCC Trivandrum 2004)

scanning, which he designed and later called as computed axial tomography scan (CAT scan) was a totally revolutionary concept in which no shadow casting was used as in conventional radiography. This image he claimed was at least 100 times more sensitive than the usual radiographic image (Fig. 25.19).

The basic work is done by the thin pencil of fan shaped X-ray beam going through the patient transversely, which are detected by complex array of sensors which detect how much the X-rays are attenuated (reduced in energy) and on basis of that they calculate the differential density of that layer. Of course the calculations involved in the construction of the image are so complex that sophisticated and fast computers are a core part of the equipment. Each square of the image calculated is termed as a Pixel or a picture element. A group of Pixels form what is called as the Voxel. The length of an voxel can vary between 1 to 20 mm and it is like the thickness of the cut in a tomographic apparatus. Each Pixel is given a number by the computer depending on the density, termed the CT number. The CT numbers vary from –1000 to +1000 and termed as the Hounsfield units.

Mandel L et al (2000)[10] reported that CT can accurately locate parotid sialoliths in patients who eventually develop sialoadenities.

Advantages of CT

- ***Different planes can be visualized***—Once the scan is complete—sagittal, coronal or transverse viewing of the body at different levels can be done by the computer without pulling up the patient again in the gantry.
- ***Greater sensitivity***—changes less than 1% can be visualized .
- Since the imaging is basically done on the transverse level the question of superimposition of anatomical structures is not an issue to confuse diagnostically.
- ***The dose of radiation*** with each generation of CT scanners is decreasing and quality of the scan is increasing. Frederiksen et al (1995)[11] have reported that the effective dose for the maxillofacial complex ranged from 0.11 mSv to 20 mSv.

Disadvantages of CT

- CT has great difficulty in imaging metallic objects anything more dense than enamel produces serious artifact giving rise to "sun ray" artifact.
- The cost of the machine as well as the procedure is very high.
- IV enhancement is necessary to show vessels.

Three-dimensional CT scan or 3D-CT is a new advance in the image analysis software. This helps in reconstructing the actual three-dimensional image of the bone and anatomical structure, including rotating it the three-dimensional axes. It helps the surgeons and diagnosticians to see the tumors in 3D details, condylar fractures, and salivary gland tumors in outstanding detail in relation to adjacent normal anatomical structures.

In many universities around the world, the input received from the computer programs is used to actual mill or carve out a 3D model out of plastic or balsam wood; using a CAD-CAM (Computer assisted design and computer assisted manufacture) machines. These models are excellent for teaching, research doing mock surgeries prior to actual ones.

With availability of the MRI imaging with better sensitivity and visualization of soft tissues of TMJ, salivary glands, the cyst and the tumors more and more diagnosticians will choose MRI where available. Cost factor can be quite prohibitive for many Indian patients who still have to pay for their own health care.

Another emerging diagnostic imaging modality is the PET scan: Myers LL et al (1998)[13] have commented upon the positron emission tomography (PET) to have an overall sensitivity, specificity, positive predictive value and accuracy of 100% in the early diagnosis of cervical lymphadenopathy associated with oral squamous cell carcinoma. PET is an functional imaging modality which was used to evaluate eleven consecutive cases of oral cancer and this result was obtained.

In India however this modality may be available only in the metros that too at exorbitant cost.

MAGNETIC RESONANCE IMAGING (MRI)

The concept of MRI is based on non-ionizing radiation imaging. It has following advantages:

- It offers the best resolution of tissues of low inherent contrast.
- No ionizing radiation is involved in MRI.
- Because the region of the body imaged in MRI is controlled electronically, direct multiplanar imaging is possible without reorienting the patient.

Basic Principle

The hydrogen nuclei behave like spinning magnets which wobble in the earths magnetic field. This is termed as precession. This precession frequency is unique for each type of nucleus. This is also termed as the Larmor Frequency.

Magnetic fields are measured in units termed as the tesla(T). One tesla is equal to the 10,000 times of normal earths magnetic field. In MRI machines the field strength of the magnetic fields varies between 0.15 to 1.5 T.

The patient is placed in a powerful magnetic field. Pulsed specific radiofrequency waves cause the nuclei to tilt away from the magnetic field. Once the radio waves are turned off, the hydrogen nuclei will return to their preferred alignment with the magnetic field and give off minute radiofrequency signals on their own. MRI is the process of locating these individual nuclei radio signals in three-dimensions and creating an image from their relative signal intensities.

These intensities depends on 3 parameters:

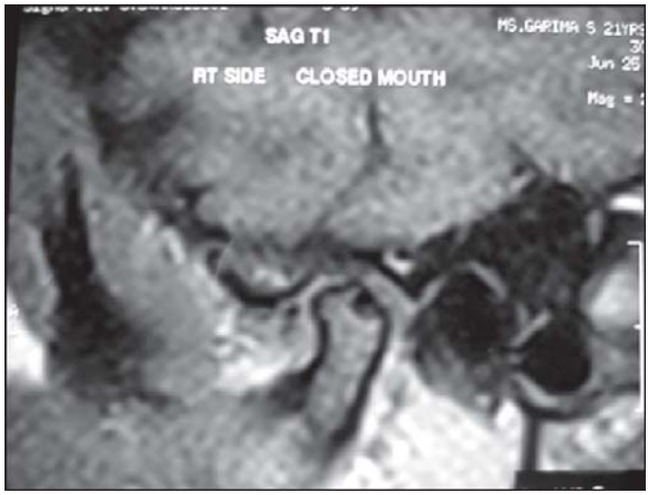
A. Proton density and nuclear motion; in other words the number of resonating hydrogen nuclei. More loosely bound are the hydrogen nuclei, more detectable will be the signal.
B. T1 relaxation time: (also known as spin-lattice relaxation time) which measures the interaction between the hydrogen nuclei and the magnetic field.
C. T2 relaxation time: (also known as spin-spin relaxation time) which measures the interaction between adjacent hydrogen nuclei.

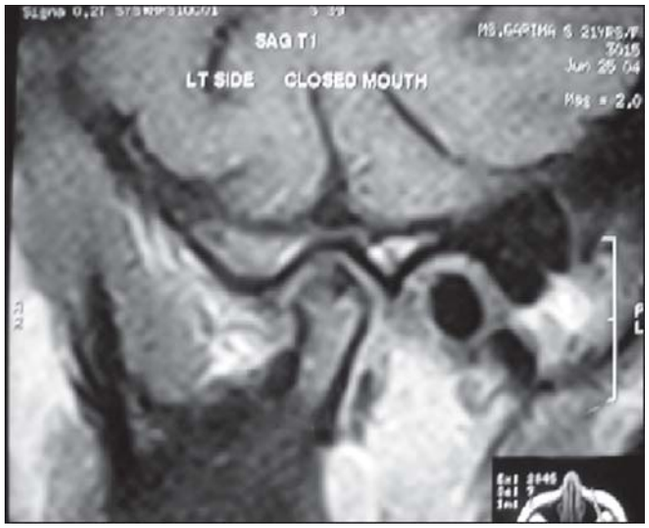
The relative T1 and T2 parameters may be adjusted to emphasize different tissues.

Generally if anatomic information is required T1-weighted image is used. Here tissues with short T1 relaxation time will produce high intensity, which is displayed as white and *vice versa*. It is also called as "fat images" because fat has the shortest T1 relaxation time. But if some one requires information on fluids or edema a T2 –weighted images are taken. Here a tissue with long T2 relaxation time will produce high intensity signal and appear bright on the image and *vice versa*. It is also called as "water images" as water has the longest T2 relaxation time.

Like CT scan, once the MR information is in the computer any number of image planes may be created. Some examples where MRI has been used for helping the diagnosticians:

- Combination of MRI with sialography is a very sensitive tool- Jager et al (2000)[4] reported that magnetic resonance sialography allows delineation of the submandibular ductal system, and detection of sialoliths with accuracy greater than ultrasound.





FIGURES 25.20A and B: Figure showing open and closed MRI image of TMJ (Sham Kishor, Bailoor DN 2004, Yenepoya Dental College Hospital, Mangalore)

- Spread of malignancies can be evaluated with great sensitivity, Gualdi GF et al (1997)[12] mention that MRI allows multiplanar studies with the advantage of a more sensitive evaluation of the extension of the neoplasms of oropharynx, oral cavity and salivary glands.
- In internal derangements of the TMJ finding the disc position is sometimes imperative for the clinician. Takebayashi S et al (1997)[14] did an unique study on 50 TMJs in which he showed a method for unique visualization of the TMJ disc. They recommend intravenous administration of gadopenetate dimeglumine (Gd-DTPA) and subsequent MRI imaging and found it more sensitive than MRI only They used 0.5 Tesla MR imager and T1-weighted images were generated (Fig. 25.20).

Disadvantages of MRI

- Relatively long imaging times.
- Contraindicated in patients with pacemaker, cerebral aneurysm clips or implants.
- Relatively expensive and unavailable in many areas in India.

Potential Hazards Associated with MRI

- ***Auditory effects of noise***: Arise from the vibration in the gradient coil and other parts of the scanner due to the varying magnetic field. Noise may read up to 95 dB causing temporary or permanent hearing loss.
- ***Claustrophobia***: This is seen in many of the patient, they feel claustrophobic inside the MRI scan. Such patients should be counseled before and may require medication.
- Magnetic field may cause ***minor physiological changes in the length of cardiac cycle***; change in red cell morphology, alteration in hemostases, increased nerve cell excitability, alteration in growth pattern. None of these are of serious considering the benefit versus risk profile of MRI.
- Magnetophosphens are visual flashes seen by subjects due to direct stimulation of the optic pathway.

ENDOSCOPIC EXAMINATION

The advances in fiberoptic imaging and miniaturization of the digital imaging cameras and videos have resulted in number of clinician using these thin tube of fiberoptics to see into various body cavities and ductal systems.

Usually the name of the endoscopes varies according to its use. One used for salivary gland visualization would be called sialoendoscope, one for joints would arthroscope and one for esophagus would be esophagoscope, etc.

Sialoendoscopy for Salivary Gland Disease Treatment

Nahlieli O et al (1997)[15] utilized a endoscope measuring 2.0-2.5 mm and inserted it into an incision of parotid and submandibular duct. They directly visualized the sialoliths and then used suction or forceps to remove them. This is a very efficient and minimally invasive method of treatment. They treated 45 cases with 80% success rate and no post-operative complication.

Nahlieli O and Baruchin AM (2000)[18] used a third generation sialoendoscope named after the researcher as Nahlieli Sialoendoscope® made by Karl Storz Tuttlingen of Germany and did a retrospective study of 236 patients whom this group has treated from 1994 to 1999 and they had phenomenal success with sialolithiasis, sialoadenitis and other forms of ductal disease. It is their contention that sialoendoscopy is a very promising method of the future for treatment of salivary gland disorders.

Video Attached to a Endoscope

Pentax videoenteroscope® — Perez-Cuadrado E et al (1997)[16] evaluated 16 patients with various abdominal problems in whom 8 were confirmed cases of Crohn's disease clinically, they found that use of push-type Pentax videoenteroscope® inserted orally was a very useful procedure for selecting the site of biopsy as well as making video documentation of the intestinal lesions.

Ziegler CM et al (1999)[17] used a videoendoscope for detection of radiolucent calculi and for therapy of sialostenosis and removal of sialoliths as well.

Endoscope used in TMJ Internals Derangements for Lavage and Lysis

The internal derangement of the TMJ with static disk was treated in 30 TMJs by Casares G et al (1999)[19] using arthroscopic treatment and they further used MRI to confirm the findings postoperatively. In this one it was possible to do lavage and lysis of the fibrous tissue and put the patient on active physiotherapy immediately. Their experience with this modality was very satisfying for both doctors and patients and they recommend it strongly.

Endoscopes will become finer and finer and recording the images using video and other digital methods will become cheaper and this interface of technology with diagnostic and treatment sciences will become routine in coming years.

RADIONUCLIDE DIAGNOSIS (RND)

The artificial manufacture of gamma-ray emitting isotopes like gallium 67 or technetium 99m and the development of

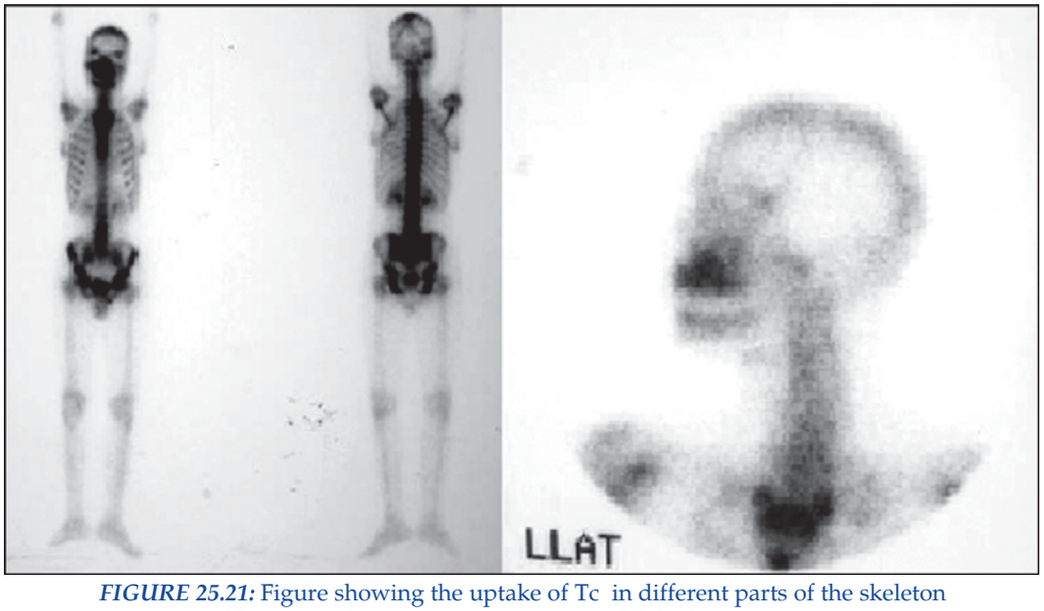


FIGURE 25.21: Figure showing the uptake of Tc in different parts of the skeleton
(Courtesy: Umarji Hemant, Ani John GDC Mumbai 2004)

Gamma scintillation camera has made the diagnostic modality of radionuclide diagnosis a reality (Fig. 25.21).

Scintillation crystal used in the Anger® gamma camera converts the gamma radiation from the patient into visible specks of light. These are amplified and converted into images into a photo-multiplier tube. Most of the images are recorded on the computer and time phase studies also use video for recording.

This modality allows for dynamic visualization of basic uptake and secretion from many of the tissues of the human body.

Technetium 99m is the preferred isotope because:

a. Gamma rays that it produces are easily detected.
b. It has a short half-life of 6 hours.
c. The residual radioactivity is negligible within 24 hours of injection reducing the side effects of the radiation damage.
d. It is biologically inert and possible to inject intravenously.

A rectilinear scanner (old) or a Gamma camera (later versions) records all of the gamma emissions from the area of interest. These gamma emissions are converted to an image that can be seen on an X-ray film or video monitor. Besides technetium 99m, gallium 67, selenium 75, and iodine131 have been used for radioactive labeling in salivary gland imaging.

The imaging is done under 3 phases of uptake.

1. ***Dynamic phase***: Spread of radioactive marker through the vascular system.
2. ***Static phase***: Concentration in the gland.
3. ***Secretory phase***: Secretion of the marker by the gland.

RND technique can be used to detect areas of increased metabolism termed as hot spot or areas of decreased metabolism like cystic areas are termed as cold spot.

Detection of metastatic tumors, salivary gland pathology, TMJ tumors and lymphatic pathologies are some of the application of this technique.

Scintigraphic Technique

Scintigraphic technique can be used for detection of salivary gland disorders. This procedure is called *sialoscintigraphy*. For this technetium 99m pertechnetate is used. This is injected to the artery and salivary gland, which rapidly metabolize will preferentially bind to it.

So after injection of the marker a set of radiographs are taken in the first 30-120 seconds. This is for the dynamic phase, followed by radiographs every 10 min for 30-45 min; this is for the static phase, finally are the patient is given sialogouge the final set of radiographs are taken for secretory phase.

Scintigraphy is much more sensitive to early or small changes in salivary metabolism than other imaging techniques. It has been successfully used to image a wide variety of salivary gland disorders including sarcoidosis, Sjögren's syndrome, sialadenitis, salivary gland tumors, Lymphomas, and postoperative healing. Also provides valuable information concerning the functional capacity of the salivary gland in patients who have undergone radiotherapy or are suspected of having either acute or chronic sialadenitis, salivary gland aplasia or sialolithiasis.

Disadvantages of Scintigraphy

a. It cannot resolve lesions smaller than 1 cm.
b. Ductal obstruction can trap the radionuclide tracer and cause a distorted image.
c. Sophisticated paraphernalia of Eluting column of Cow radioactive element, expensive Anger® gamma camera and ofcourse the most important the trained diagnostician who can make sense of all these hot and cold spots.
d. A single scan of technetium 99m can result in full body radiation of 1mGy which is equivalent to 33% of the annual radiation that one gets on average from the background radiation in nature.

San Pedro EC et al (1995)[5] reported that sialoscintigraphy is a simple noninvasive procedure that can usually separate benign entities like Warthins tumor from malignant tumor. Vigh L et al (1997)[20] have introduced two functional concepts in the radionuclide diagnosis, the TUI or Total uptake index and the SSGR Stimulated salivary gland response used as a diagnostic tool. Both the index and SSGR had lower values in Sjögren's syndrome.

Another recent advance includes an imaging modality, which combines the nuclear medicine concepts with computerized tomography concepts. Most investigators claim that these scans can produce three-dimensional

images 100 times more sensitive than the RND scans. This has been called the PET scan or positron emission tomography scan. The radionuclides it uses are, $O^{15,}$ F^{18} etc. PET requires on site cyclotron to produce these short-lived radioactive molecules.

None of the dental schools in India have access to such sophisticated modalities but one must learn where the future is going. What is science fiction today will become reality in our life time, much sooner than expected.

CONCLUSION

Good knowledge of the anatomy will guide the radiographer to modify the techniques to an individualized mode of X-ray visualization. Svenson B et al (1996)[1] has recommended use of E-speed film and rectangular collimation as means of reducing the radiation dose in practice. They strongly recommend that continuing dental education programs are a must to sensitize the dental practitioners to the latest information on radiation protection.

The specialized techniques need the doctors to be trained properly for their optimal use and moreover the cost factor is still very prohibitive one as far as its use to common man is concerned. Less than 10% of the population can get access to these tertiary hospitals. This means that for a long time many primary care dentists will have to depend on the greatest tools given to man- empathy, caring and a sharp diagnostic mind.

It is well said, "*A fool with a latest tool, is still a fool*"!

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Bailoor DN, Pai Keerthilatha

26 Films and Media

INTRODUCTION

Historically starting from the glass X-ray plates to cellulose to now polyester plastic media have been improved continually over this century for better image and reduction in dose. The computers heralded a paradigm shift in the media with the charge coupled device and phosphor storage media dominating the imaging scene. The Xeroradiography made a brief appearance on the maxillofacial radiology scene but it was to become obsolete due to cost and awkwardness of processing. Today almost all the media we can look forward to will be digital. Days are not far when the dark room, and the processing chemicals will be kept in dental museum with the Howard Raper's Radiology textbook.

BRIEF HISTORICAL REVIEW

Early 1900s Glass plates wrapped in black paper prone to crack and break

1913 Hand wrapped cellulose nitrate films—They were inflammable

1924 Cellulose triacetate films—wrinkle and distort

1960 Polyester base (Dacron) has been used since

1980 Digital radiography—Research and development

1990 CCD based imaging gains routine acceptance

2000 Only digital imaging—Multiimaging facility of thermography, NMR, CT scan time giving immediate help to diagnostician, this will be backed up with expert systems—computer assisted medical decision making.

Images will be teleported all over the hospital through Networking and all over the globe through wide area networks (WAN) using satellite technology, fused with multimedia for teaching, and stored on optical discs for long times, easily cross referenced and instantly retrieved.

STRUCTURE OF FILM

The film: The X-ray film consists of two components.

- *Emulsion*
- *Base*

The *emulsion* is made up of Silver Halide crystals (Silver bromide and silver iodide) and the gelatin matrix for support. Sulphur is added by manufactures as a contaminant to increase radiosensitivity of the emulsion. *Base* is made up of polyester-polyethylene terephthalate which is flexible and translucent.

The ekta speed films (E speed) have a marking EKT and only the E speed films must be used in the clinics today since they allow good radiographic visualization with minimum radiation exposure. At the corner of each

dental film is a dot, which is raised towards the side of exposure. By convention in our department we keep the dot towards the occlusal side (i.e down in maxillary and up in the mandibular) This helps to tell the side of the radiographed area easily. Towards the concave side of the dot is the lead foil and the film should not be exposed from that side. Periapical film comes in three sizes.

Size	0	Pediatric film	————	22 × 35 mm
Size	1	Adult anterior	————	24 × 40 mm
Size	2	Standard adult	————	32 × 41 mm

FIGURE 26.1: Showing extraoral and intraoral films used usually in the dental departments. All the names appearing on the products are trademarks of the respective company (Bailoor DN, Keerthilatha Pai, 2004)

In your dental clinic the same films may be used for bitewing projections by making a paper tab on your own or readymade tabs are also available commercially.

An *Occlusal* film is usually 57 × 76 mm in size. (Or 2.5 inches × 3 inches in size). The non-screen film is usually adequate for most of the routine dental work.

Extraoral films come in two main types those that are compatible with regular screens and those that are used with rare earth screens. The sizes of extraoral films could range as follows:

5 × 7″, 8 × 10″, 10 × 12″ and 12 × 12″

If special sizes are required they may be cut in the dark room from the stock 12 × 12″ packets.

The *orthopantomographic* films come in 5 × 12″ and 6 × 12″ with suitable cassettes and rare earth screens (Fig. 26.1).

Intensifying Screens

- Definition
- Structure
- Types

The intensifying screens are those radiographic ancillaries, which use the principle of fluorescence to obtain a better image with lesser amount of X-ray exposure.

Structure—Base-Polyester plastic

Reflecting layer—Titanium dioxide—helps in reflecting any Light from the fluorescent layer.

Fluorescent layer—It may contain Ca tungstate,

Terbium activated - Gadolinium oxysulfide.
Thulium activated - Lanthanum oxybromide.

Protective layer of plastic

The above mentioned crystals produce visible light on exposure to the X-ray (calcium tungstate or the other two components) and this principle of generation of visible light due to action of X-ray photons is termed as Fluorescence. The films are also suitably designed in their chemical structure to be more sensitive to specific color of the spectrum. Thus it is important to follow manufacturer's instruction as for the selection of compatible film for each screen. Logically the screens are placed inside the cassette in close juxta position to the film.

Types

We classify the calcium tungstate screens as regular and other two, i.e. gadolinium and lanthanum as rare earth. The rare earths have only one disadvantage in the Indian context, they are very expensive (Fig. 26.2).

Processing X-ray Film

The processing of X-ray films is divided into two types: manual and automatic

- The Manual is further of two types-
  - The time—temperature method and the visual method

FIGURES 26.2A and B: Showing intensifying screens of calcium tungstate variety used in the department of maxillofacial radiology at the Yenepoya Dental College and Hospital Mangalore (Prasanna Kumar, Bailoor DN 2004)

- The chart suggested for time-temperature method is suggested time temperature chart to be fixed near the timer and developing tank

Temperature	*Development time*
76°F	3- 3.5 minutes
72°F	4- 4.5 minutes
70°F	4.5- 5 minutes

- The range is given since the new and slightly used solutions developing solutions will require little more time.

Processing basically involves putting the exposed film through the developing solutions, washing with water, then dipping in fixing solution for fixed amount of time, then to washing, drying and storing it for reference.

Detailed Explanation

When the X-ray film is exposed to the information—carrying beam of photons coming out of an object, the photosensitive silver halide crystals in the film—emulsion that interact with these photons are chemically changed. These chemically altered crystals are said to constitute the latent (invisible) image on the film. The concept of the latent image implies that chemical changes produced by the X-ray increases the ability of the altered crystals to the chemical action of the ("developing") process that converts the latent image to visible image. Knowledge of processing principles is also necessary to anticipate and prevent many of the pitfalls inherent in this multi-step procedure.

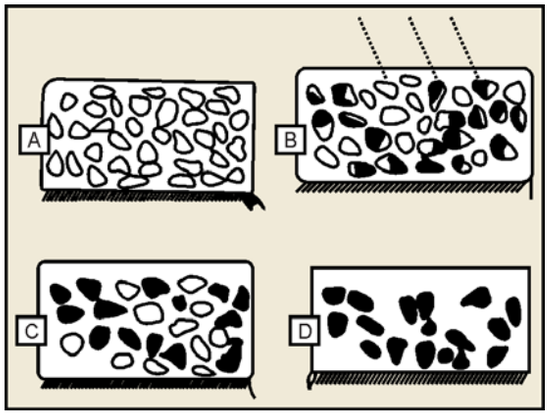
Formation of Latent Image

The film emulsion is a suspension of tiny photosensitive silver bromide and silver iodide crystals that have been precipitated in gelatin and layered to a thin sheet of transparent plastics base. These silver halide crystals are imperfect in several ways.

They contain a few free silver ions in the spaces between the crystalline lattice positions. These are referred to as interstitial silver ions. There are physical distortions in the regular rectangular array of the silver and bromide ions in the crystals due to the presence of the iodine atoms occupying some of the bromide sites. The silver halide crystals are chemically sensitized by the presence of (added) sulfur compounds play a critical role in image formation and, as with the physical irregularities in the crystal produced by the iodide ions, are called the latent image sites.

Their function is to begin the process of image formation by trapping the electrons generated when the emulsion is irradiated. There are many such latent image sites in each crystal.

When the silver halide crystals are irradiated, X-ray photons interact primarily with the bromide ions by Compton and photoelectric interactions. These result in the removal of an electron from the bromide ions with the



FIGURES 26.3A to D: Above showing A: Unexposed crystals of silver halide. B: Exposed film with latent image is evident. C: Developer releases the metallic silver on the film D. The unexposed silver halide is removed by hypo or fixer (Nillofer Shabnam, Bailoor DN Yenepoya Dental College and Hospital, Mangalore)

production of high speed electrons and scattered photons. By the loss of the (recoil) electrons the bromide ions are converted to bromine atoms that are absorbed by the gelatin of the emulsion. The recoil electrons move through the crystal generating additional bromine atoms, secondary recoil electrons and scattered photons until a major portion of their energy has been expended and they encounter a latent image site. Here they become "trapped" and thereby impart a negative charge to the site. The positively charged free interstitial silver ions are attracted to the negatively charged latent image site. When the silver ion reaches the charged latent image site it is neutralized, with the result that an atom of metallic silver is deposited at the site. This process occurs many times over at a single site within a crystal whenever photons and recoil electron strike bromide ions. After exposure of a film to radiation, the aggregate of silver atoms at the latent image sites comprises the latent image. It is the metallic silver at each latent image site that catalyzes the development of the halide crystal in which it is formed, that is, renders the crystal sensitive to development and image information. The larger the aggregate of silver atoms the more sensitive the crystal is to the effects of the developer. The primary actions of the processing solution are to convert the crystals with the latent images to black metallic silver grains that can be visualized and to remove the unexposed silver bromide crystals (Fig. 26.3).

Processing Solutions

Film processing involves the following procedures:

- Immersion of exposed films in developer solution.
- Rinsing in running water.
- Immersion in fixing solution.
- Film washing.
- Drying and mounting for viewing.

Developer Solution

The action of the developer is to reduce all the silver ions in the exposed crystals of silver bromide (with a latent image) to metallic silver. This reduction process must be restricted to those crystals containing a latent image in order to produce a diagnostic image. For this purpose, the reducing agents used as developers are those that are catalyzed by the presence of the metallic silver at the latent image sites. The metallic silver at the latent image sites appear to act as a bridge by which the electrons from the developing solution can reach the silver ions in the crystal and convert them to metallic silver. Individual crystals are developed completely or not at all during the recommended developing times. Variation in densities on the processed radiographs are the result of uneven distribution of developed (exposed) and undeveloped (unexposed) crystals in the areas. Those areas that have a larger proportion of exposed crystals will be the more dense (blacker) because of the higher concentration of black metallic silver granules in the areas after development. If the developer is permitted to remain in prolonged contact with silver bromide crystals that do not contain a latent image, it will slowly reduce them also and thereby 'overdevelop' the image.

When an exposed film is being developed there is an initial period in which no visible effect of the developer is apparent. After this initial phase the density increase, very rapidly at first, then more slowly. Eventually a time is reached at which the exposed crystals are all developed (reduced to black metallic silver) and the unexposed crystals are beginning to be reduced by the developing agent. Reduction of unexposed crystals results in the production of chemical fog on the film. This interval between maximum density and fogging explains why a properly exposed film does not become "overdeveloped"

even though it may be in contact with the developer longer than the recommended interval. Thus dark films are usually the results of overexposure, not over development. It should also be noted that an overexposed film will develop larger more effective latent image sites, which explains why such a film will develop acceptable density in a shorter period of time than a film exposed for the proper (shorter) time. Such a circumstance results in increased exposure of the patient and is to be discouraged.

The developing solutions contain four components:

i. Developer
ii. Preservative
iii. Activator
iv. Restrainer

- ***Developer***: The primary function of the developing agents is to amplify the latent image by covering the exposed silver halide crystals into metallic silver grains. This process is initiated at the latent image where the electrons from the developing agents are conducted into the silver halide crystal interior and reduce constituent silver ions to metallic silver. Unexposed crystal without latent images are unaffected during the time allowed for the reduction of the exposed crystals. This emphasizes the importance of carefully controlling the time for development. To critically control the developing process, two developing agents are usually present in the developing solutions used in dental radiology:
  - Elon (monomethyl-para-amino-phenol sulfate)
  - Hydroquinone (para-dihydroxy benzene).

  The hydroquinone brings out the contrast of the image. It is quite sensitive to temperature changes, becoming inactive below 60°F and very reactive above 70°F. Thus the temperature of the developing solution is critical. Elon is less temperature sensitive and acts quickly to bring out the gray shades in an image. This combination of Elon and hydroquinone is widely used in photographic developers for use with film and photographic paper.
- ***Preservative***: The developing solution contains a preservative, usually sodium sulfite, which has a great affinity for oxygen. The addition of the preservative helps protect the developers from being oxidized by atmospheric oxygen.
- ***Activator***: The developers are only active at high pH values, usually above pH 11. To maintain this condition, the developing solution contains alkali, which serves as an activator. The alkalies generally used are sodium carbonate, sodium hydroxide, and sometimes sodium metaborate and tetraborate. The activators also serve to soften the gelatin so the developer agents can diffuse more rapidly into the emulsion and reach the silver bromide crystals suspended in the gelatin.
- ***Restrainer***: The fourth component added to the developing solution is a restrainer, usually potassium bromide. The bromide is added because bromide is a product of the reduction of the silver halide crystals and the added bromide serves to depress the reduction by the common ion effect. It is much more effective in depressing and acts as an antifog agent.
- ***Developer replenisher***: Developer replenisher is a solution made for topping off the developing solution each morning. It is a more concentrated solution of the developer constituents designed to replace those depleted from the originally used solution without unduly increasing the volume. Its high pH will compensate for the reduced alkalinity of the used solution and will partially offset the inhibitory effect of the accumulating bromine atoms, a major factor limiting the life of the developing solution.

Rinsing in Running Water

After development of a film the emulsion is swelled and saturated with the developing chemicals. At this time the films should be rinsed in water for 15 to 20 seconds before they are placed in the fixer. This rinse dilutes the developer and thereby slows the development process. The rinse also removes the alkali activator, thus preventing neutralization of the acid fixer.

Fixing Solution

The function of the fixing solution is to remove (dissolve) the undeveloped silver halide crystals from the emulsion. The presence of the unexposed crystals from the emulsion. The presence of the unexposed crystals cause film to be opaque, so if these crystals are not removed, the image on the resultant radiograph is obscured and nondiagnostic.

A second function of the fixing solution is to harden the emulsion on the film.

The fixing solution also contains four components:

i. Clearing agent
ii. Acidifier
iii. Preservative
iv. Hardener

- ***Clearing agent***: Sodium or ammonium thiosulphate removes the unexposed silver halide from the emulsion, the clearing agent normally does not affect the silver grains deposited on the emulsion during the development, but excessive fixing may dissolve the grains reducing density of the film.
- ***Acidifier***: Acetic acid is added to the fixing solution to rapidly neutralize any developer which is alkaline in nature. It preserves the pH below 6 so that action of fixing solution can take place efficiently.
- ***Preservative***: Sodium sulphite—It helps to prevent the decomposition of the hypo (Thiosulphate clearing agent) from the solution.
- ***Hardner***: Aluminium potassium sulphate hardens the gelatin so that it can stand handling storage.

Processing

In the time—temperature method

72° F ———————— 4 minutes
76° F ———————— 3 minutes

The above mentioned combination appears to be ideal. But in most of the dental clinics—the visual method is used in which the radiographer sees the partially processed dental film in the safe light till he has optically seen the images or visualizes the pulp chamber—Then washes and fixes for a minimum of 5-6 minutes. The films are again washed and then mounted on the commercially available mounts prior to interpretation. It is customary in India to mount with the raised dot at the corner of the film towards the observer.

Automatic Processors

In the dental practice we recommend an automatic processor with daylight loaders, the processing chemistry is same and the only difference is that a roller system transports the film through developer, fixer, water and drying chamber. 4-6 minutes are required to drying of film. Then this method takes out the drudgery of the film processing (Figs 26.4 and 26.5).

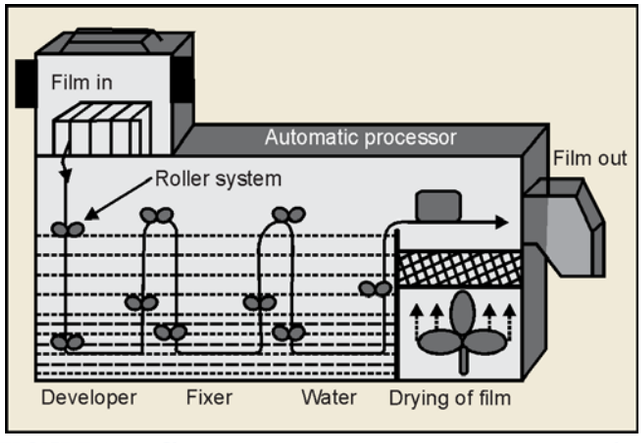


FIGURE 26.4: Showing a automatic process in use at the Yenepoya Dental College and Hosptial, Mangalore (Sonali Bailoor, Bailoor DN, 2004)

FIGURES 26.5A and B: Showing a automatic processor in use at the Yenepoya Dental College and Hospital, Mangalore (Sonali Bailoor, Bailoor DN, 2004)

Rapid Processing Chemicals

They are now available which usually take 10-30 seconds to process the film—Extremely useful in sports dentistry, endodontics and emergencies—The contrast is sub optional and this method is not advocated for routine processing.

The following are:

Radiographic image characteristics:

- *Radiographic density*—It is the overall degree of darkening of the dental X-ray film.
  Graphic plotting of film density to the exposure is termed as the characteristic curve or also the H-D curve or Hurter and Drif-field curve after the pioneer investigators.
- *Radiographic contrast*—It is the difference in the densities between the various regions of the film.
- *Latitude*—It is the range of contrasts that may be recorded usefully.
- *Radiographic Mottle*—Radiographic mottle or noise refers to the appearance of uneven density of an unexposed film.
- *Sharpness*—The ability of the radiograph to precisely define an edge of a change in the density of the object visualized.
- Area covered by the radiograph should also be correct and as per the request of the clinician otherwise even with all the above characteristics being optimal it will not be a quality radiograph.

Grids

Grid is defined as that radiographic accessory which when placed between the patient and the film, as close as possible to the latter, helps in reducing the scattered radiation.[2]

The scattered radiation is that which emanates from the object being radiographed and is dependent on the object thickness, size of the collimator and the kVp used. This scatter is usually twice to four times the intensity of primary beam.

The grid is made up of alternate layers of radiolucent, i.e plastics and radiopaque, i.e lead which are aligned in the direction of the primary beam either parallel (unfocussed) to each other or at an angle (focused). In general grid has 80 line pairs per inch.

Grid ratio—The ratio of the thickness of the grid to the distance between the spacer is termed as the grid ratio.

The moving grid is normally used to get rid of the radiopaque fine lines that may appear on the radiograph. It is also termed as the Potter-Bucky diaphragm.

Most of the extraoral radiographic projections of the skull like PNS views, Caldwell view, Submentovertex view are best visualized using grids with screen films.

Uses—It helps to reduce the film fog and improves the contrast.

DIGITAL RADIOGRAPHY OR DIGITAL IMAGING (RADIOVISIOGRAPHY RVG)

Four main topics will be touched upon here

- Direct digital radiography
- Indirect digital radiography
- Digitization of the existing radiographs
- Digital subtraction radiography DSR
- Selenium based digital radiography systems

Direct digital radiography uses the CCD or the charge coupled device, which converts the radiographic image into analog signal that in turn is taken up by the software loaded in the computer that makes the image visible. The image can then be enhanced using the programming in the software, many of which are able to generate colored images having increased sensitivity (Fig. 26.6).

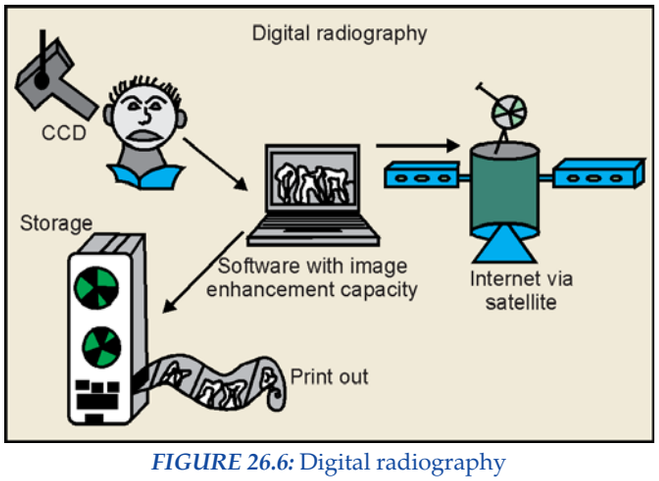


FIGURE 26.6: Digital radiography

Disadvantages of the RVG

1. Currently available digital radiography equipment for periapical techniques have resolution of 11 line pairs per mm, whereas the silver halide Ekta speed film has slightly more than 20 line pairs per mm resolution.
2. Cost is still quite excessive and in most smaller town such an equipment will not pay for itself.

Advantages of RVG

a. The image is instantly available—no dark room delay
b. Coloring of the image can highlight slight changes in the densities.
c. Radiation dose is more than halved for most views.
d. The image can be sent over the hospital intranets or local area networks.
e. The image can be electronically packaged and sent over the internet.

The panoramic machine is also now fitted with the CCD sensor and the computer shows the ortho-pantomographic image on the screen. Nessi R et al (1998)[6] have evaluated the digital panoramic radiography and proved it to be a useful tool to study metal implants after maxillofacial surgery as well as alveolar bone structure. The dose reduction is very important to young patients who may undergo repeated radiographic examination and follow up in implant procedures.

Scarfe WC et al (1997)[7] have used the RVG radiography with niobium filtration and found that there was 42 percent reduction in the dose as compared with the E-speed film. But the *in vitro* studies using standard phantom heads found that soft tissues such as thyroid received increased doses. Most workers are working with every project to decrease the dose received by the patients.

Indirect Digital Radiography

Phosphor storage screens—Special phosphor storage screens are used in many of the digital systems and then a special laser reader is used to generate the image which is stored in the computer hard disk. It can be similarly compressed and sent over communication lines.

Digitization of Existing Radiographs

The existing X-rays can be digitized using special scanner in transparency mode or photographs can be made of radiographs and then scanned in using a low end scanner which does not have a transparency setting.

Digital Subtraction Radiography (DSR)

This is an specialized digital technique in which two radiographic digital images (Some time apart, like after 4 weeks or 4 months, etc) of the same region made by standardized methods are made and then the two images are superimposed. Special software is used to subtract the regions that are unchanged and thereby highlighting those regions where there is a difference.

Some Examples

Three digital radiographs of a diabetic patient made one year apart will show how the periodontal bone loss progresses. Periapical cyst after endodontic surgery and follow-up of few weeks will show how bone heals in the DSR pictures. Most DSR images can be viewed on the computer console and CCD techniques are the best for their visualization.

Reddy MS (1997)[3] mentions that techniques like digital subtraction radiography permit detection of bony changes too small to be seen by the unaided eye and are over 95% sensitive and specific in detecting bony changes.

Nummikoski P V et al (2000)[4] concur that DSR is a digital radiographic image subtraction method designed to enhance detection of crestal or periapical bone density changes and efficiency of a good image editing software will be really helpful in improving the diagnostic ability.

Selenium based Digital Radiographic Systems

Ludwig K et al (2000)[5] have worked with selenium based digital radiography and compared the diagnostic performance with that of regular film and phosphor based laser reader systems(Digora®) and found that these new systems hold a promise for even further reduction of the radiation dosage.

Xeroradiography

Xeroradiography uses the recording media of selenium plate.[1] This plate is a semiconductor which retains the surface charge on it. By virtue of this property, it is able to retain the image of the plate after discharging with X-ray beam.

This plate is then put in a processing chamber where it is sprayed with toner particles and these bring out the image. A white photographic quality white paper is pressed on to this plate and covered immediately with a transparent tape that protects the toner consisting of fine black particles from getting smudged. The image is viewed in reflected light and does not deteriorate with time.

The soft tissue and hard tissue visualization is excellent in this method and is still the method of choice for early detection of breast pathology. The radiation to the oral tissues is also much less than the conventional Ekta speed films.

Xeroradiographic cephalometrics has been richly quoted in literature since the soft tissue profile and the tongue position is sharply visualized. With the CT scans and MRI now available this modality will never be seen more in dental institutions.

This method never really caught on in Indian Dental Institutions because of the high cost and only marginally better images. At the same time the CCD based detectors became available which had better imaging qualities and lesser cost.

Today Xeroradiography is one the dinosaurs of the dental imaging world which suddenly became extinct !!

CONCLUSION

The radiographic examination whether it is intraoral periapical technique or panoramic radiographic technique the digital CCD direct methods are reducing the doses and new methods of filtration like Niobium are being investigated to further reduce the dose to ALARA levels.

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27 Radiographic Interpretation

INTRODUCTION

The proper interpretation of the radiograph remains both an art and science. An art to be learnt under the guidance of experienced teachers and science to master the fundamentals of the anatomic landmarks, different lesions and their differential diagnosis. Increasingly the diagnosticians may be asked to make diagnosis on the computer screen with the universal appeal of the digital radiography (Fig. 27.1).

FIGURE 27.1: Radiographic investigations help in Scientific evaluation of bony lesions Omal P.M, Bailoor D.N 2004 YDC)

SYSTEMATIC APPROACH

The diagnostic sequence:

a. Accurate history and thorough clinical examination
b. Clinical impression
c. Selection of the type of the radiograph and its proper execution
d. Proper mounting and viewing
e. Describe the lesion seen
f. Enumerate the differential diagnosis
g. Integrate the other investigations—histopathology-complete blood analysis, etc.
h. Arrive at definitive diagnosis
i. Initiate the treatment plan.

Step 1: The clinician should keep in mind the history, the clinical examination and hold a tentative diagnosis in the mind prior to prescribing the radiographic view. If the suspected lesion is involving one or two teeth then go for intraoral periapical radiograph; If the lesion is larger like a 3″ × 3″ swelling on the palate then choose a occlusal radiograph ; If you have a large slow growing swelling affecting the whole angle of the mandible then obvious first choice would be a panoramic view.

Step 2: Proper processing, mounting (correctly orient the left and right) and then viewing in optimal conditions (In a room which has low ambient lighting). Use of a magnifying loupe is highly recommended.

Step 3: Review existing radiographs if any with the new films.

Step 4: Look at the normal landmarks in all its variants and then locate the abnormal site or lesion on the radiograph.

Step 5: Radiographic description starts with the view taken, of the side being described, of the site, size, shape, symmetry, borders and content. Relate the associations. Giving a scientific opinion of what the descriptors mean in clinical sense.

Step 6: Many times it's not possible to arrive at single diagnosis then a list of probable differential diagnosis is made. When additional evidence is obtained from microscopy, serology or hematology a final decision is made as to the treatment planning.

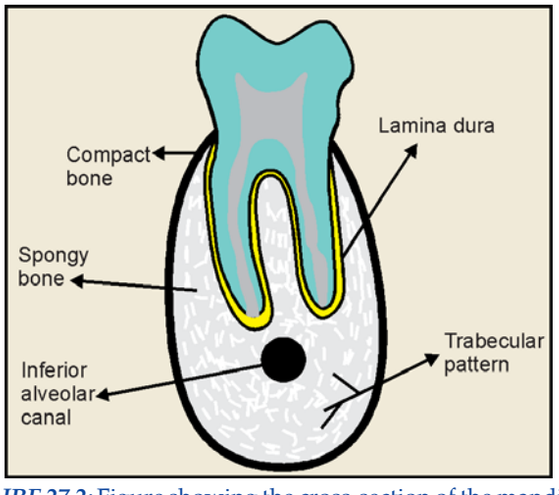


FIGURE 27.2: Figure showing the cross-section of the mandible depicting the different types of tissues encountered by intraoral periapical radiographic pictures. The compact bone and lamina dura is radiopaque, trabecular pattern is slightly radiopaque with more radiopaque lines representing trabeculae. The enamel is the most radiopaque density on the film, second comes dentin and almost radiolucent is the pulp chamber (Prasanna Kumar, Bailoor DN 2004, Yenepoya Dental College Hospital, Mangalore)

Petrikowski CG et al (1996)[4] commented that radiography is one area where there is considerable inter-observer variability and he attributes it to clinical bias, education, training, and experience.

The understanding of the normal landmarks of maxilla and mandible are very important to the primary aspects of the radiographic assessment (Fig. 27.2).

Radiographic interpretation is a science, in itself. Correct interpretations of radiographs require a thorough knowledge of basic anatomical landmarks. One patient may not show all the normal anatomical landmarks in one radiograph. This does not mean that these landmarks do not exist, but only that the angulation of X-radiation and other factors changes their appearance on the radiograph. Knowledge of appearances of normal landmarks is mandatory, only then can a novice dental surgeon differentiate these with the appearances of various pathological lesions.

There are many normal anatomical landmarks radiolucent and radiopaque in mandible and maxilla; though present together, for convenience we study separately.

RADIOLUCENT LANDMARKS OF MAXILLA

- **Incisive foramen or incisal foramen or anterior palatine foramen:** It is the oral termination of nasopalatine canal, transmitting nasopalatine nerves and vessels. Present palatally at the middle of central incisors, it can be of various shapes *viz*. mere slit, rounded, oval, rhomboid, heart shaped, etc. Average size is a site for incisive canal cyst. The cyst causes enlargement of the foramen upto a size of around 1 cm, its presence over the central incisors may be mistaken for periapical lesions. The point of differentiation can be:
  - Intact lamina dura
  - Tooth will be vital
  - Shadow will shift from its original position by the change in horizontal angulation for the X-rays in cases of normal appearance.
- **Intermaxillary suture:** Also known as median palatine suture. This appears between two portions of the pre-maxilla as a thin radiolucent line between the center of roots of incisors. This is visible usually in young children. It appears as a dark line extending from central incisors to the posterior aspect of the palate. Width of the suture is almost uniform; Only in very young patients, it may terminate as funnel shaped widening at the anterior end. Margins are lined by cortical bone which appears radiopaque. The cortical margins may and may not be uniform.

The appearance of this radiolucent line is sometimes mistaken for fracture in the alveolar process in periapical films and fracture of the palate in occlusal films.

- **Nasal fossae or nostrils:** These appear as dark shadows over the lateral incisors. The nasal, cavities are air filled, therefore, appear as radiolucent areas in periapical radiographs of anterior teeth. These shadows are quite dark because of air in them. Nasal septum, a dark radiopaque line, divide the two fossae. The margins of the fossae are lined with compact bone. Therefore in radiograph the dark shadow of the cavities are lined with narrow white lines (Fig. 27.3).

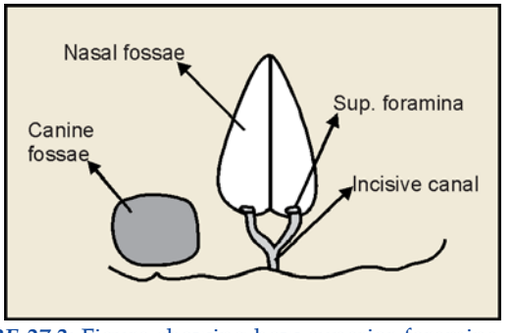


FIGURE 27.3: Figure showing how superior foramina merge into incisor canal. Canine fossa Radiolucency is very diffuse. (Prasanna Kumar, Bailoor DN 2004 YDC)

- **Nasopalatine Canals:** This is usually not seen in periapical film but can be viewed in Occlusal films. This canal originates at two foramina at the floor of the nasal cavity. The openings are on either side of the nasal septum (Fig. 27.4).

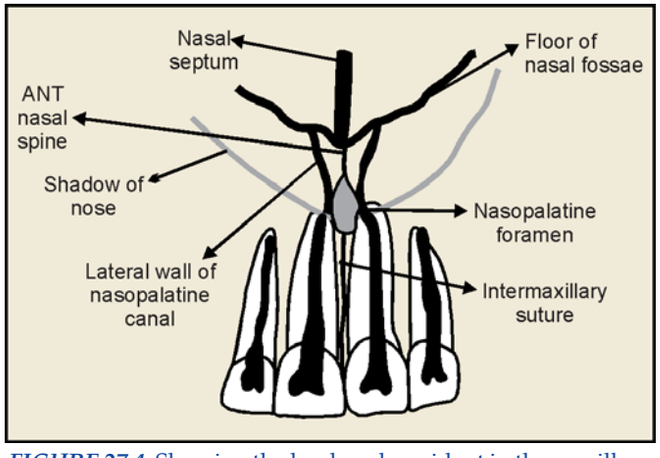
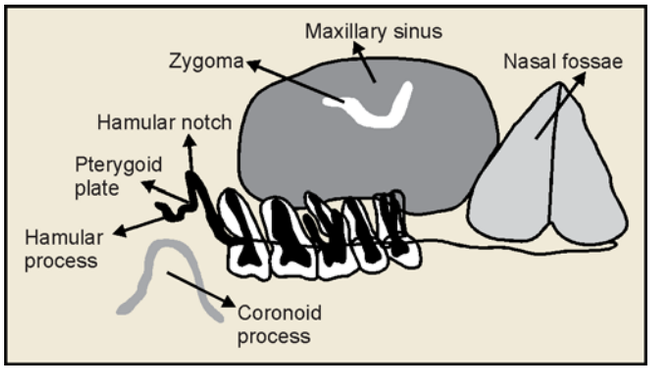
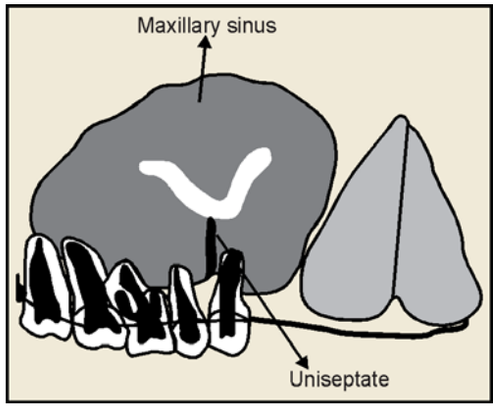
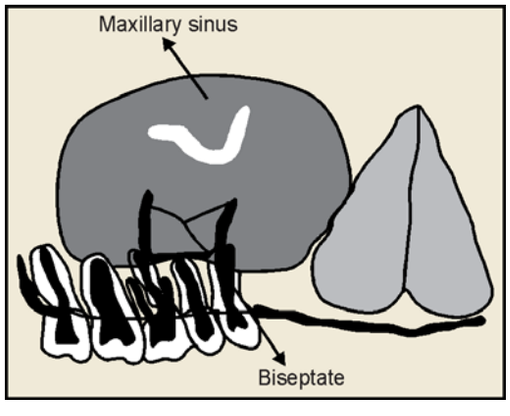


FIGURE 27.4: Showing the landmarks evident in the maxillary anterior IOPA. All landmarks are not evident in all films. (Prasanna Kumar , Bailoor DN 2004, Yenepoya Dental College and Hospital, Mangalore)

- **Antrum of highmore or maxillary sinus:** This appears as dark shadows over the posterior teeth usually from premolar to the tuberosity region. This appears quite dark because it contains air. Maxillary sinus is the largest of the paranasal sinuses. The two sinuses right and left can be of similar shape or different[1] (Fig. 27.5).







FIGURES 27.5A to C: Showing the single, uniseptate and bi-septate maxillary sinus (Prasanna Kumar, Bailoor DN 2004; Yenepoya Dental College and Hospital, Mangalore)

On the intraoral periapical radiograph it appears as either U shaped or W shaped with one septa or rarely with two or more septae. In the IOPA there is always U shaped radiopacity which is the shadow of the zygoma. It is also termed as malar process by some authors.

Sometimes, the maxillary sinus exhibits uniform shadows of nutrient canals. They can follow any directions, usually the course is convex towards the alveolar process.

- **Nasolacrimal duct:** This is seen in occlusal films and very rarely in periapical films. This is round or oval shaped radiolucent area over the roots of the first molar. It can be slightly mesial or distal to it. This can be superimposed over the apices of either second bicuspid or first and second molar (Fig. 27.6).

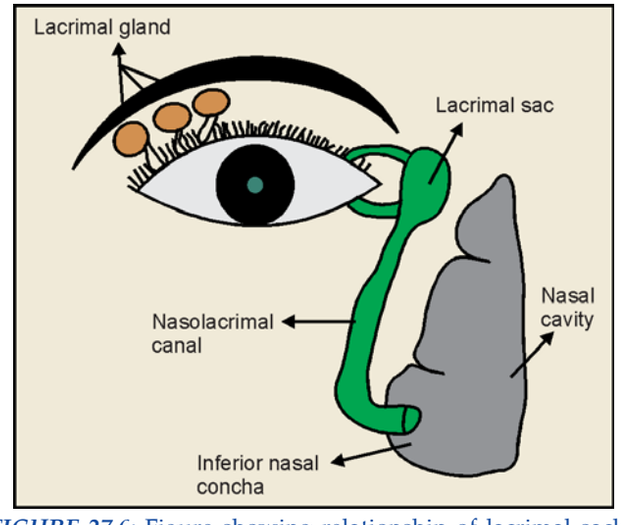
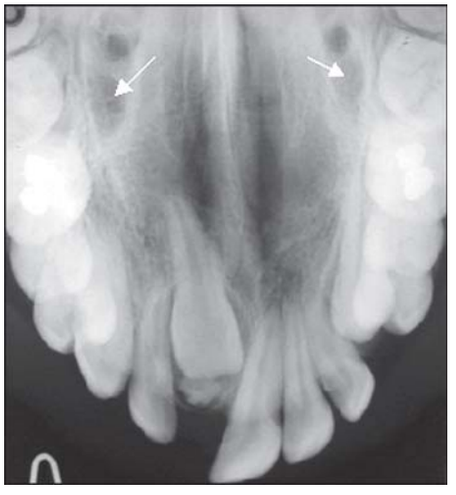
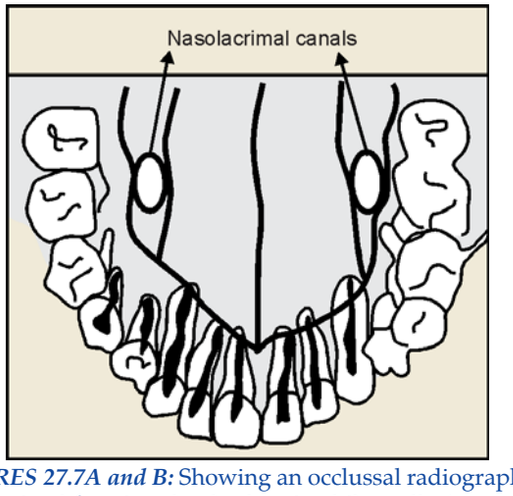
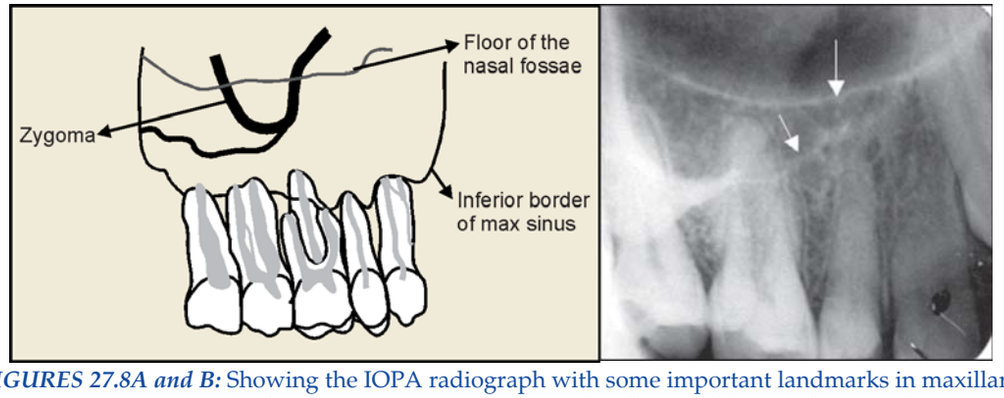


FIGURE 27.6: Figure showing relationship of lacrimal sack, nasolacrimal canal and its drainage into inferior nasal concha. (Prasanna Kumar, Bailoor DN, 2004 YDC)





FIGURES 27.7A and B: Showing an occlussal radiograph with nasolacrimal formina clearly showing bilaterally (see arrows) (Courtesy: Varghese Mani, GDC, Calicut)



FIGURES 27.8A and B: Showing the IOPA radiograph with some important landmarks in maxillary posterior region (Bailoor DN 2004 Yenepoya Dental College and Hospital, Mangalore)

- **Posterior palatine foramen:** This is seen only in occlusal films and very rarely in periapical films. This is a round or oval shaped radiolucent area over the roots of the first molar.
- **Median palatine suture:** Seen in occlusal films, a thin radioluscent line in the centre of the palate.

RADIOPAQUE LANDMARKS OF MAXILLA (Figs 27.7 and 27.8)

- **Zygomatic process and the bone:** In the periapical radiograph, the zygomatic process appears as a U-shaped radiopaque line with its open end directed superiorly. It is seen often in the maxillary sinus radiolucency.
- **Zygoma or malar bone:** It appears as an irregular radiopaque shadow covering the third molar apices which may extend up to the apices of second molars. In cases where palatal vault is low, this shadow of malar bone may be misinterpreted as hypercementosis or as ankylosis of second and third molars.
- **Hamular process or sphenoid bone:** This is seldom visible in intraoral films. In extraoral films this appears as a thick radiopaque line terminating just below the region of maxillary tuberosity.
- **Nasal septum:** It is seen as a pear shaped radiopaque area extending backwards from the incisive foramen in between two central incisors.

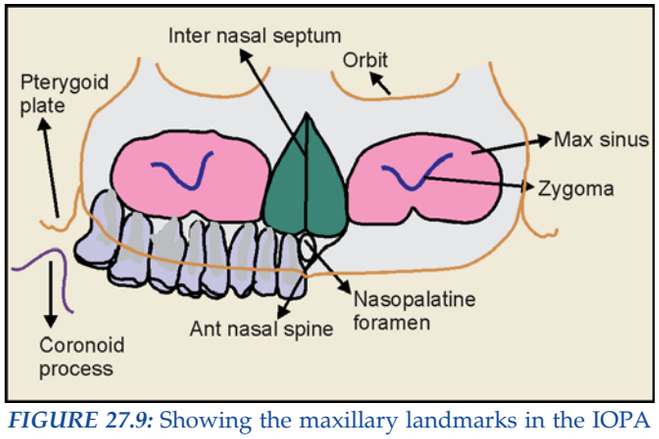


FIGURE 27.9: Showing the maxillary landmarks in the IOPA radiographs. Diagrammatic representation helps to understand and locate the landmarks and distinguish them from pathology (Bailoor DN, Prasanna Kumar 2004, Yenepoya Dental College and Hospital, Mangalore)

- **Inverted 'Y' of Ennis:** In the periapical region of the maxillary canine IOPA radiograph the lateral wall of the nasal fossa and the anterior border of the maxillary sinus forms an inverted Y which is termed as "inverted Y of Ennis" after one of the senior researchers in oral radiology Dr Ennis.
- **Coronoid process of mandible:** It is a triangular gray area of radiopacity seen on the radiograph of upper molars (Fig. 27.9).

RADIOLUCENT LANDMARKS OF MANDIBLE

- **Mental foramen:** It is present under the roots of first and second premolar. It is visible mostly in youngsters because as the age increases, the mental canal is directed superiorly and posteriorly. The shape of the foramen may vary from round to oblong. The size can vary from 1mm to 0.5 cm. Usually it is corticated.

  Presence of intact lamina dura and testing the vitality of the tooth can distinguish it from the pathology. By change of angulation of cone slightly the foramen will move while the periapical pathosis will remain with the root apex.
- **Mandibular foramen:** This is only visible in lateral jaw films. As a small rounded or funnel shaped black shadow over the ramus of mandible.
- **Mandibular canal:** It commences from mandibular foramen in the ascending ramus. This appears as a radiolucent area covered superiorly and inferiorly by

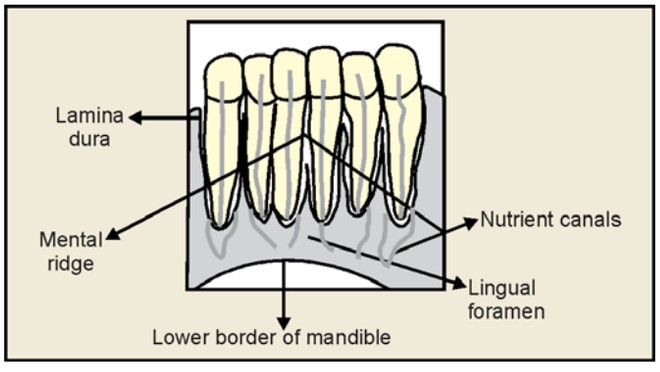


FIGURE 27.10: Showing the diagrammatic representation of mandibular anterior IOPA to understand the normal landmarks. (Prasanna Kumar, Bailoor DN 2004, Yenepoya Dental College Hospital, Mangalore)

radiopaque margin. Borders are seen partially or even may be absent. Position of canal varies; canal lies below the roots of the molars and little distance below the bicuspids. The apices of the molars may appear to be superimposed over the canals. Anatomically, the canal lies buccal to the molars and premolars. The width of the canal varies from 3 mm to 1 cm (Fig. 27.11).

- **Mental fossa:** The mental fossa is a depression found on the labial aspect of the mandible on the anterior side. This may be mistaken for the periapical pathology of the mandibular anterior teeth.
- **Nutrient canals or interdental canals:** These are often seen in mandibular periapical radiographs. They carry neurovascular bundle in the jaw bones and supply the teeth and gingival tissues. Width of nutrient canal may vary from 100 micron to 1mm. Margins of the canal may reveal a thin white cortical plate which may be slightly irregular (Fig. 27.10).
- **Pharyngeal space:** This is seen as a radiolucent area, only in lateral jaw films, as a broad dark area extending vertically on ramus. It is caused by patients swallowing when the film is being exposed. Care must be taken to see the area and to differential diagnose it with pathological lesions.
- **Submandibular gland radiolucency:** The body of the angle is thinned down physiologically in most of the cases. The thinness which appears free from the regular trabecular patterns must be differentiated from the pathologies like osteoporosis, fibrous dysplasia, ameloblastoma, etc. also termed as submandibular gland fossa.

RADIOPAQUE LANDMARKS OF MANDIBLE (Fig. 27.12)

- **External oblique ridge:** White line on the anterior portion of ascending ramus. Sometimes it is so heavy that it is overshadows the roots of the molars.
- **Genial tubercle:** These are four in number, two on either side of the median line on internal surface of mandibular incisors. This is usually seen in occlusal films. This appears as a white ring with a dark centre immediately beneath and between lower central incisors.

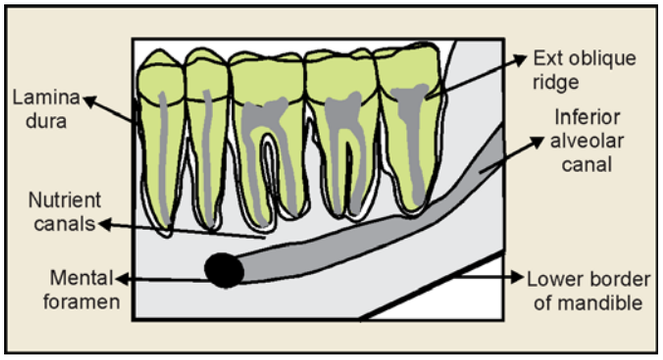


FIGURE 27.11: Showing different landmarks seen in the mandibular posterior IOPA (Prasanna Kumar, Bailoor DN 2004 Yenepoya Dental College and Hospital, Mangalore)

- **Mental ridge:** A dark white ridge extending from symphysis to the bicuspid region. Some times superimposed by apices of lower anterior teeth.
- **Mylohoid ridge:** This appears as a white line starting from lower border of symphysis and continuing upwards in the molar region towards ramus. Some times overlaps the molar apices.
- **Border of mandible:** This appears as a heavy white line on the radiograph. This is seen on IOPA whenever increased negative angulation is given in mandibular posterior radiography.

Above all, the shadows of teeth and pulp cavities appear as radiopaque and radiolucent respectively in both maxilla and mandible. The radiopacity of enamel, dentin and cementum varies but the radiolucency of

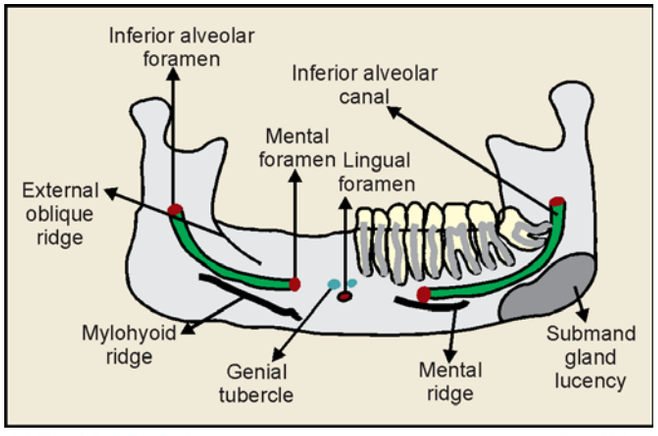


FIGURE 27.12: Showing mandibular landmarks in IOPA radiographs (Bailoor DN, Prasanna Kumar 2004, Yenepoya Dental College Hospital, Mangalore)

pulp cavities is uniform. Pulp stones appear radiopaque.

RADIOGRAPHIC APPEARANCES OF PATHOLOGICAL LESIONS[2]

After having studied anatomical landmarks, it is important to keep in mind the appearances of some common lesions occurring in the jaws with typical findings.

- **Radiographic appearance of caries:** Caries, in very simple terms, can be classified into occlusal caries, proximal caries, buccal/lingual caries and cemental caries. The caries usually progress along the enamel rods. Here only the radiographic appearance will be stressed upon.
- **Occlusal caries:** Initial occlusal caries is not seen on a IOPA radiograph. It is better detected by explorer and dental mirror. As it progresses and reaches the dentinoenamel junction it becomes apparent as a radiolucent triangular area with base towards the DE junction.

  Later on the radiolucent area becomes bigger and approaches the pulp, but it is important to remember that this is a two-dimensional picture of a three-dimensional object. Consequently it may mislead the observer into thinking that the pulp is involved when actually it is only buccal or lingual extension.

  It is best not to commit from the radiograph about the pulpal involvement in initial cases but to depend on clinical features.
- **Buccal and/or lingual caries:** As mentioned above, these caries also present as radiolucent areas, oval or semilunar in shape and often give an optical illusion of pulpal involvement even when actually the caries is far from affecting the pulp horn.
- **Proximal caries:** The initial or incipient caries starting in the interdental areas results in focal spots of radiolucency which rapidly spreads into bigger oval and is best visualized in the bitewing radiographic technique, as compared with IOPA technique.

  The secondary caries or the caries which begins under the restorations, or crowns is also best visualized by the BW technique.
- **Cemental caries:** This type of caries are more common in geriatric dental patients where recession has occurred.

  Radiologically these appear as saucer shaped lesions. They can appear on any tooth surfaces, but mesial surfaces are more susceptible.

  These lesions are to be differentially diagnosed from caries under the class II preparations and even erosions at these surfaces. The neck of the tooth, area between crown and the root, absorb less X-ray and appear radiolucent, either uniform all around or more consistent on the proximal surfaces. This is known as 'cervical burn out'. Careful examination of the patient clinically along with careful 'reading' of the radiographs is important. In certain other cases, restored teeth appear radiolucent because the filling material is radiolucent.

Radiographic Appearances of Periapical Lesions

Following lesions need to be differentiated from each other

- Discontinuity of the lamina dura
- Periapical granuloma
- Periapical cyst
- Periapical abscess
- Periapical cemental dysplasia-cementoma
- Endodontic scar
- Teeth with incomplete formation of roots
- Orthodontically moving teeth show small lesions which heal on completion of the treatment
- Rare lesions

The radiographic appearance of the periapical lesions vary greatly.

- First sign would be the loss of intact lamina dura.
- The second is the small bulb like radiolucent lesion in a non-vital tooth with a sharp reactive border of bone formation-granuloma.
- Third is a rounded or oval shaped radiolucency more than 5 mm in diameter usually in contact with a non-vital tooth.
- Fourth is an irregular bordered or hazy bordered radiolucency in a tooth which is painful on vertical percussion.
- Fifth is a benign tumor of the cemental tissues and belongs to the group of lesion termed as the cementoma. It is seen in three stages.

I. Fully radiolucent stage
II. Mixed radiolucent and radiopaque specks stage
III. Completely radiopaque blob of pathologic cementum stuck to the root periapex, tooth is vital and non-symptomatic.

Stages I - III takes about 12-18 months.

Sixth, history of the recent endodontic surgery will normally alert the clinician to the possibility of the postoperative scar. Rare lesions discussion will not be attempted in this section.

Root Resorption

Root resorption is usually pathological but physiological root resorption during shedding of deciduous teeth do occur. Physiological root resorption always occurs uniformly. Pathological root resorption can be internal or external

Internal root resorption occurs as a result of local irritation with the resulting inflammation of pulp. Any trauma resulting in disturbance of circulation of pulp, may lead to resorption of the tooth. This process, if continues, may lead to perforation of the root. Root resorption after pulpotomy operation has been reported. Occasionally deciduous molars show internal resorption.

External root resorption usually involves permanent dentition. These can be due to:

- Inflammation of periodontal ligament resulting from trauma or certain periapical diseases due to pulp canal infections.
- May be because of pressure of impacted tooth.
- Along side of tumors or cysts.
- Idiopathic root resorption is also common.
- Root resorption after reimplantation is quite common.
- Concommittant internal and external root resorption can occur.

Hypercementosis

It is an excessive deposition of cementum either along the entire length of root or only in apical region. It may be due to systemic effects or because of infection so mild which causes constructive rather than destructive activity.

Radiographic Appearance of Periodontal Diseases

Periodontal disease, being the most prevalent, radiographic appearances are important. Following check up of the patients with bitewing radiographs is mandatory to know the initiation of periodontal disease.[3]

- The first sign, thickening of periodontal space can be registered.
- Periodontal ligament thickening in maxillary premolars is due to trauma from occlusion.
- The very initial sign that periodontal lesion has set in is the loss of crestal bone in the interdental area.
- The horizontal bone loss is that which shows the resorption in the interdental area (radiolucency) which is parallel to the line joining the two cementoenamel junctions.
- Angular bone loss or vertical bone loss is that which the radiolucency of the resorbed bone causes the acute angle to form, or the angle between this and the CE junction is acute or sharp. Normally suggestive of primary trauma from occlusion or secondary trauma from occlusion.
- The radiolucency which is oval shaped surrounding the cervical areas of the tooth are indicative usually of the periodontal abscess, of course the pain on lateral percussion and the clinical signs of periodontal abscess are seen in it.

Full mouth bitewing radiography is best to evaluate serially the initial progress of periodontal disease. Other lesions like the cyst, the benign tumors, TMJ diseases and the paranasal sinus diseases, etc have been described in the various chapters. To the student of oral radiographic interpretation; a bible of interpretation "HM Worth's—Oral Radiographic Interpretation" is highly recommended. Of course the real teacher of the practicing dental surgeon will be his clinics and his input from the experience.

CONCLUSION

Interpretation of dental and orofacial radiographs is truly an art and slowly with experience one learns to unzip the secrets held in the blacks and whites of the radiographic image.

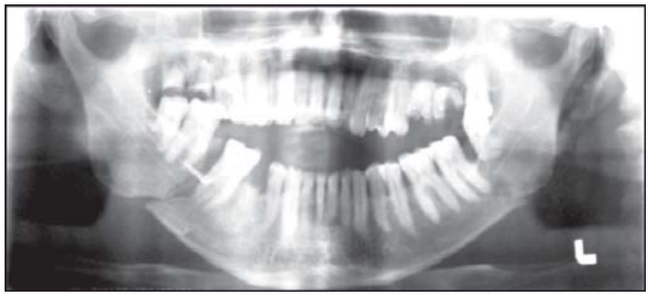
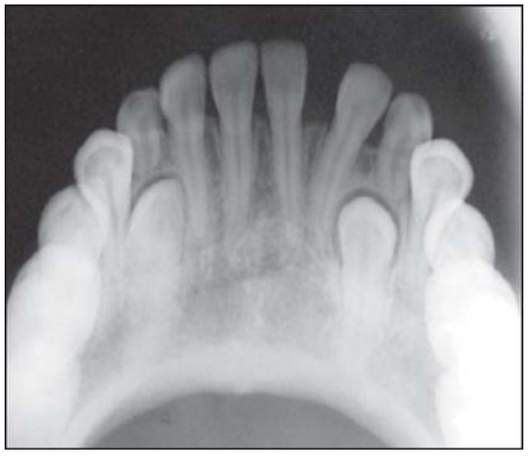


FIGURE 27.13: A clinician must request OPG when he feels that the pathology could be wide spread like in a road traffic accident. Here we notice that two areas show the fracture, both angle of mandible regions this was a 22-year-old male in bike accident where he fell sideways. (Chatra LK, Bailoor DN 2004 Yenepoya Dental College and Hospital, Mangalore)



FIGURES 27.14A and B: Showing how occlussal radiograph helps in the visualization of the whole upper or lower jaw, specially the teeth bearing areas. Usually true or topographic views are taken and these can also help in localization of objects in three-dimensions by the right angle technique (Keerthilatha Pai, Bailoor DN 2004)

FIGURE 27.15: Showing how altering the density of development of the film can help in better diagnosis of the radiating radiopaque lines at the periphery. This was diagnosed as osteogenic sarcoma and subsequently confirmed histopathologically (Courtesy Rajiv Borle, Sharad Pawar Dental College, Wardha 2004)

FIGURE 27.16: Showing lateral oblique view with massive destruction of the body of the mandible with tooth hanging in air appearance. Typical of oral cancer infiltrating into the mandible (Courtesy Ramachandran RCC Trivandrum 2004)

Knowledge of anatomical variation of the landmarks is the fundamental basis for furthering the differential diagnosis list. Any diagnosis is a three legged stool which rests on history and clinical examination; histopathological examination and the image analysis of the radiographic findings. Any written report should consider all the three aspects and then suggest the differential diagnosis (see Figs 27.13 to 27.17).

With the new image processing software, the digital images on the computer screen can even be colored and this can lend a new sensitivity to the early detection of the incipient lesions.

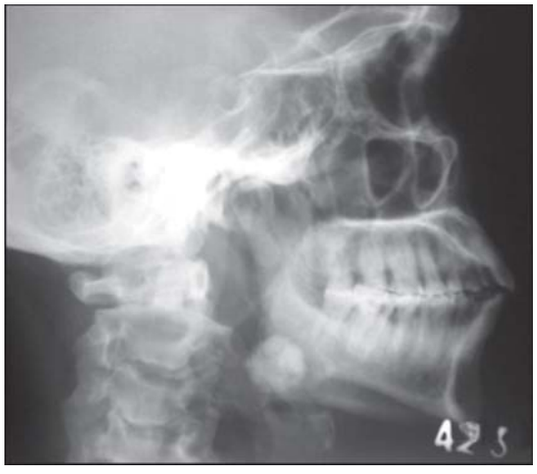


FIGURE 27.17: Showing a true lateral view of the skull with huge calcification in the submandibular salivary gland which was surgically removed (Courtesy Ramachandran RCC Trivandrum 2004)

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Chatra LK

28 Errors in Dental Radiography

INTRODUCTION

The dental student who makes radiographs at his graduate training frequently tends to make errors. This chapter intends to give him a basic framework on which to investigate these, and profit from the experience.

Density is the amount of blackness seen in the radiograph. Contrast is the level of distinction that can be made between the layers of dark and light in the projection.

Detail is the accurate reflection of the small anatomical minute in the film.

IDEAL RADIOGRAPH

The ideal radiographs is that which shows:

- Optimum density
- Optimum contrast
- Accurate detail
- Covers the area of interest completely
- It is indicated for that clinical condition.

When any of the above conditions are not satisfied it may be termed as the faulty radiographs.

FAULTY RADIOGRAPHS

The faults in the radiographs may be due to either one of the following:

1. Projection errors
2. Exposure and processing errors
3. Artifact errors

PROJECTION ERRORS

1. **Shortened image**
   - Excessive vertical angulation (bisecting angle technique)
   - Film not parallel to long axis of the tooth (paralleling technique).
2. **Elongated image**
   - Insufficient vertical angulation (bisecting angle technique)
   - Film not parallel to long axis of the tooth (paralleling technique).
3. **Overlapping of the teeth**
   - Incorrect horizontal angulation.
4. **Cone cut**
   - Improper adjustment of X-ray tube; head-cone does not cover the film completely.
5. **Apical ends of teeth cut off**
   - Film not placed deep enough
   - Insufficient vertical angulation.
6. **Blurred image**
   - Movement of the X-ray tube or patient during exposure
   - Excessive bending of the film.

7. **Double images**
   - Film exposed twice to radiation
8. **Tyre**—Track effect (herring bone effect)
   - Opposite side of film placed towards tube.

EXPOSURE AND PROCESSING ERRORS

1. **Light radiographs—Low density**
   - Exposure time low
   - kVp low
   - mA low
   - SFD too large
   - Developing temperature low
   - Exhausted developer
   - Excessive fixation.
2. **Dark radiographs—High density**
   - Exposure time high
   - kVp high
   - mA high
   - Developing time more
   - Developer temperature high
   - Developer concentration high
   - Inadequate fixation
   - Accidental exposure to light
   - Improper safe-lighting.
3. **Low contrast radiographs**
   - kVp too high
   - Under exposure
   - Under development.
4. **Film fog**
   - Improper safelighting
   - Prolonged exposure to safelight
   - Light leak
   - Over development
   - Over strength developer
   - Contaminated solutions
   - Outdated films
   - Films exposed to radiation
   - Strong temperature/humidity high.
5. **High contrast radiographs**

- Insufficient kV.

ARTIFACTS ERRORS

1. **Dark spots on radiograph**

- Film contaminated with developer before processing
- Film in contact with tank/another film during fixing
- Fingerprints (Figs 28.1 and 28.2).

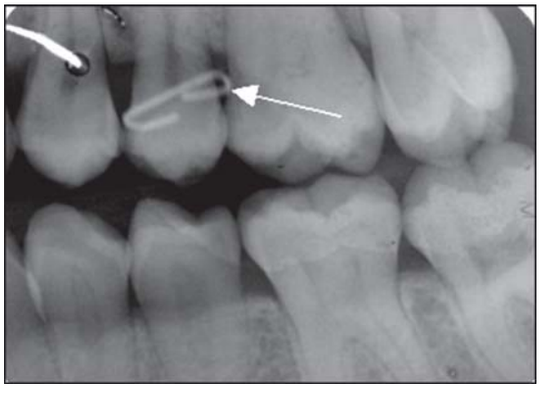
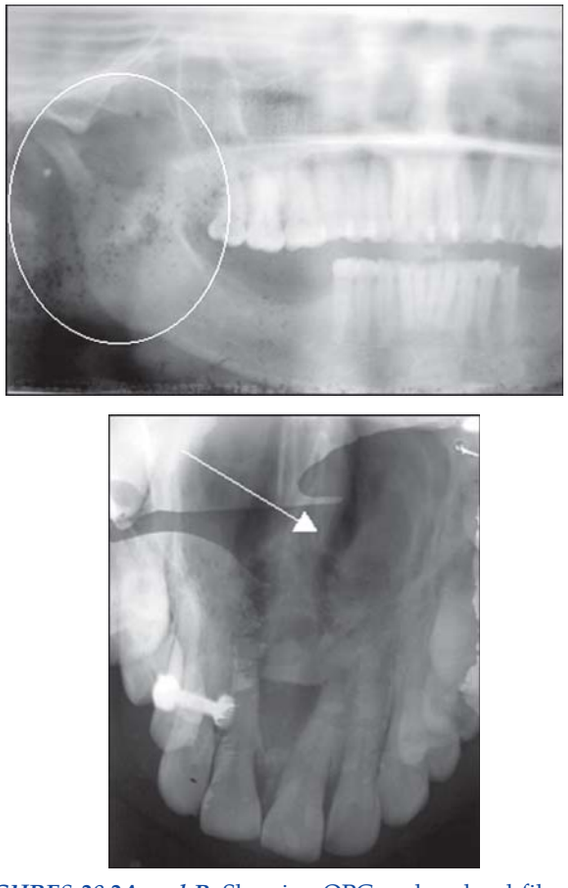
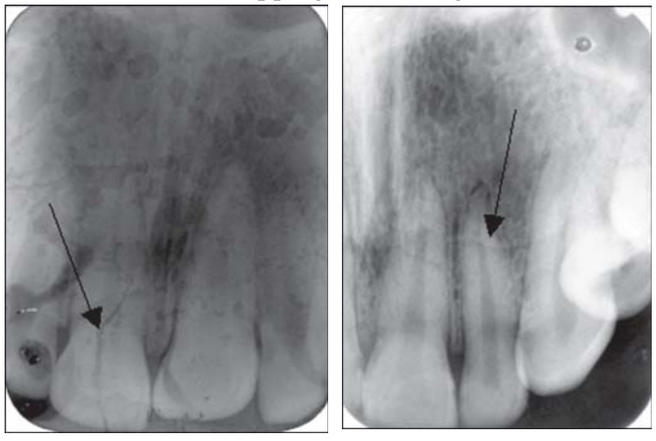


FIGURE 28.1: Showing the artifact of staple pin used as biting block for bitewing radiograph (Beena K, Omal PM, Bailoor DN 2003 Yenepoya Dental College and Hospital, Mangalore)



FIGURES 28.2A and B: Showing OPG and occlusal film that was contaminated before exposure (Prasanna K, Nillofer S, Bailoor DN 2003 Yenepoya Dental College and Hospital, Mangalore)

2. **Light spots on radiograph**
   - Film contaminated with fixer before processing
   - Film in contact with tank/another film during developing.
3. **"Nail-mark" artifacts**
   - Excessive bending of film.
4. **"Static-electricity" artifacts**
   - Forceful unwrapping of films (Fig. 28.3).



FIGURES 28.3A and B: Static electricity artifacts (Omal PM, Beena K, Bailoor DN 2003 Yenepoya Dental College and Hospital, Mangalore)

5. **Yellow/Brown stains on film**
   - Exhausted developer
   - Oxidised developer
   - Insufficient washing (Fig. 28.4)
   - Depleted fixer
   - Contaminated solutions.
6. **Blisters on film**
   - Air bubbles on film during development
   - Temperature difference developer and fixer
   - Excessive acidity of fixer.
7. **Blurring of image**
   - Movement of patient or tube head
   - Exposure twice on the same film.
8. **Ghost image (Fig. 28.5)**

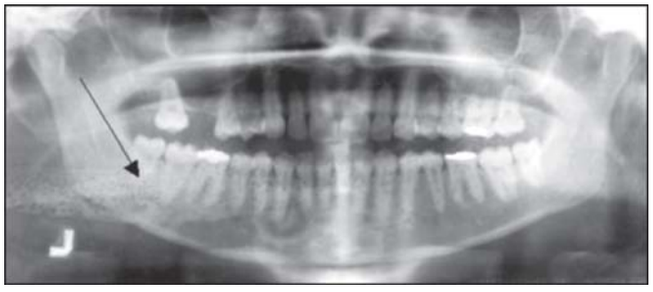


FIGURE 28.4: Figure showing yellow stain due to insufficient washing (Prasanna K, Nillofer S, Bailoor DN 2003 Yenepoya Dental College and Hospital, Mangalore)

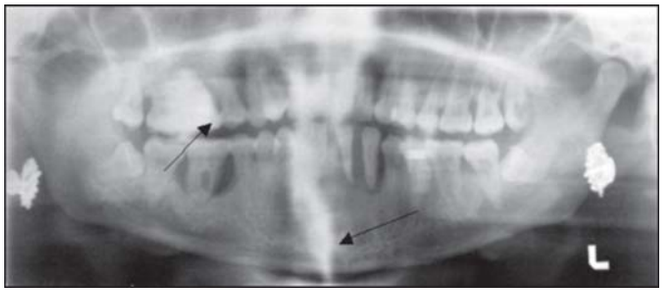


FIGURE 28.5: Ghost image (Nillofer S, Prasanna K, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

CONCLUSION

This list is neither complete nor comprehensive but a representative of the commonly occurring errors. If the dental surgeon can identify the causes of these errors and develop his own chart for troubleshooting, he will go a long way in giving his patient a quality controlled radiographic service.

FURTHER READING

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Balaji Rao B

29 Orthopantomography

INTRODUCTION

The term Pantomography is derived from the word Panorama 'an unobstructed view of a region in every direction'; Tomography the Greek word meaning "X-ray technique for making radiographs of layers of tissue in depth, without the interference of tissue above and below that level". Panoramic radiography does not replace the conventional dental film, but when used as a supplemental aid, it produces a comprehensive radiographic survey never before realized by the dentist (see Fig. 29.4).

Three different methods have been used in panoramic radiography: in one method, intraoral source of radiation is used to project on to the film positioned on the patient's face (Reverse-radiography based on Microfocus Principle) (see Fig. 29.2A), in another method, the source of radiation and the film are positioned extraorally (Rotational Panoramic Radiography) (see Fig. 29.2B) and in the third the latest CCD sensor (see Fig. 29.8) technology makes it possible to have digital images recorded in the computer and displayed on the Visual Display Unit (see Fig. 29.3).

More and more tertiary level hospitals are going for the digital and filmless imaging systems.

HISTORY

Today most of the well-equipped Dental Institutions in India have the Panoramic Radiographic Apparatus. The debt we owe to giants of science in this field is remembered by outlining briefly how time consuming and tortuous was the path to the present state of development of this unique maxillofacial radiography equipment.

Panoramic radiographs are being made using two distinct methods.

- Use of intraoral source of radiation
- Use of curved surface tomographic processes.

In **1943,** Horst Beger [16] of Dresden Germany was issued a patent to make radiographs from intraoral tubes.

In **1946,** without the knowledge of this patent Walter Ott[17] a Swiss dentist developed an dental X-ray tube that could be introduced into the mouths of the patients.

The intraoral type employs an intraoral source of radiation. The radiation is directed from inside the mouth and the film is wrapped around the patient's jaw, No screen or the Slow speed films are used. This X-ray tube must have a very small focal spot (0.10 to 0.15 mm width) tube current = 0.5-1.00 mA and kVp ranging between 40-80.

Following three companies made the modern accepted intraoral anode type of panoramic machines:

- Koch and Sterzel Co of Essen Germany—Panoramix ?
- Siemens Corp, Erlangen Germany, Status—X ?
- Philips Medical Systems Inc, Holl and (Stat Oralix)?

Rotational tomographic process was by far the most popular method whose initiation credit goes to H Numata[18] of Japan. In 1933, he was the first to propose and experiment with this method. Numata placed a curved

film lingual to the teeth and used a slit of an X-ray beam to expose the film.

1946—Yrjo Veli Paatero[19] of the Institute of Dentistry, University of Helsinki, Finland refined this technique and finally gave it the name of Parabolography. In 1950, he termed this technique as Pantomography.

1938—Watson of England read a paper on tomography, which stated that film used need not always, be flat, and was considered an heretic in the existing tomographers.

Milwee (1937) and Heckmann (1939) are credited with what they termed as the slit scannography of curved surfaces.

1950—Nelson RJ and Kumpula JW worked at University of Washington at Seattle and developed technique of radiography similar to one developed earlier. Then Paatero was invited to work at the University of Washington and by fall of 1951 another prototype was designed. Following are some important land marks in the development of the OPG—

1954—Blackman S of England collaborated with Paatero and developed a commercially viable Rotograph.

1956—Hudson D and Kumpula file a US patent for their panoramic machine.

1958—Sairenji E of the Nihon University Japan suggested to Paatero the name of Orthopantomography.

1961—Paatero was made the chairman of the Oral Radiology at the University of Turku, at Finland.

1962—Faude J of SS White developed a machine with a chair shift.

1979—Orthopantomograph 5 was introduced.

1980—Morris CR San Antonio Texas. Panorex II

1984—Orthopantomograph 10 customized the focal trough by three plane light line system.

Many companies have come out with digital OPG machines of these we have shown only Promax from the Planmeca OY (See Fig. 29.7), Finlands machine as an example whose details were readily available to us on request.

PANTOMOGRAPHIC MACHINE (See Fig. 29.1)

Pantomography provides a sharp image of a selected curved tissue plane. Shadow superimposition is reduced by a special radiographic technique. The tube and film rotates and the film is exposed by a narrow X-ray beam; and film moves at a speed that follows the moving projection of a certain object point.

The machine is designed to produce a panoramic flat picture of the jaw arches by means of a narrow rotating beam and a moving film. The apparatus contains X-ray source; a cassette holder; a small tube slit; a positioning guide for the jaws and a mechanical coupler between film and the cassette.

Working principle foci of projection in the vertical and horizontal plane are not the same. The functional focus is constituted by the X-ray source in the vertical plane and rotation center of the beam in the horizontal plane.

FIGURE 29.1: Showing the Xtropan 2000 ® Indian made OPG machine manufactured in Sewree West Mumbai, India

- Principle of layer formation when a beam of a finite width scans an object, the image is successively exposed on a stationary film. The projection of an object point moves at the film plane as the beam passes the point. The object moves in the same direction as the beam movement but at a lower speed. If the film moves at a speed that follows the moving projection at a certain object point, this point will always be projected on the same spot on the film and will not appear unsharp in

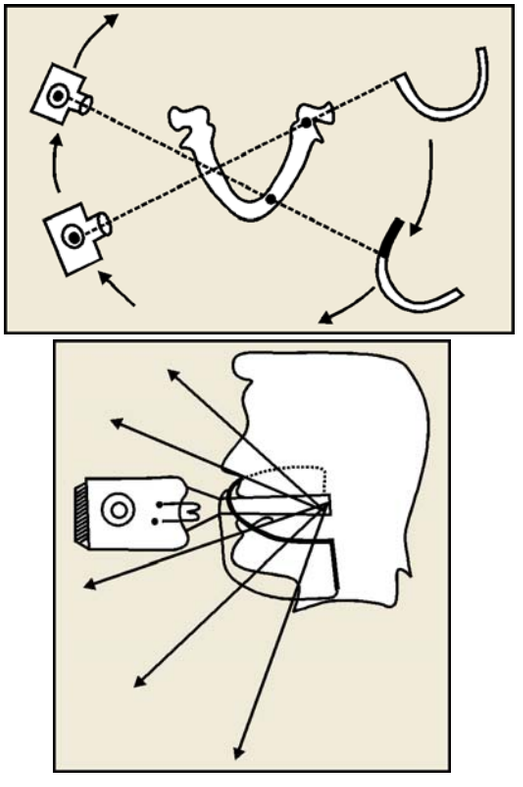
the resulting image. In rotational panoramic radiography the film is attached to the rotating system and moves in the same direction as the beam. The film is given the correct speed by opposing the movement with a contrary movement relative to the beam.

The projection of object points outside the sharply depicted plane, either towards the rotation center of the beam or towards the film, will have a different projection speed at the film plane than the film itself, and will be blurred out. In this way a zone in the object may be defined as that which contain those object points that are depicted with sufficient resolution so that they may be distinguished, which is referred to as image layer.

- Form and thickness of image layer: A constant film speed in relation to the beam places the center of the image layer at a defined distance from the rotation center of the beam. If the speed of the film is not constant during the exposure, a continuous shift of the position of the layer takes place. Acceleration shifts the position of the image layer successively away from the rotation center of the beam and deceleration shifts the position of the layer successively towards the rotation center.[8]

  The distance from the rotation center of the beam to the center of the image layer is called the effective projection radius. The thickness of the image layer is dependent on the length of this (effective projection) radius. The longer the radius, thicker the image layer, and is inversely proportional to width of the beam.
- Practical application of the working principle since the jaw is not circular, variety of different movement patterns for the beam have been developed by manufacturers. The simplest projection technique applied in practice utilizes one stationary rotation center of the beam placed at one side of the jaw projecting the other side of the jaw on to the film. One side of the jaw is exposed at time and the position of the rotation center, is shifted symmetrically between the exposures by moving the patient. This projection technique creates the so called 'split image'. [8]

  In systems creating "Continuous Images", several different movement patterns of the beam are utilized to achieve the desired projection of the jaws. The objective is to project each part of the jaw as close to perpendicular as possible. Two basically different techniques can be distinguished:
- The utilization of several combined stationary rotation centers of the beam,
- The utilization of continuously moving rotation center. Some may use a combination of both.
- Stationary rotation centers to produce a continuous image, a combination of three stationary rotation centers may be used. During exposure, the beam is first rotated around a laterally positioned rotation center, which serves as a functional focus while the major part of the opposite side is exposed. When the scanning approaches the anterior region the rotation center of the beam momentarily shifts to the midline position posterior to the dental arch and the anterior region is exposed with second rotation center as the functional focus.



FIGURES 29.2A and B: A depicting the rotational curved surface tomography with curved cassette and B showing the panoramic machine working on reverse radiographic projection principle. Here the X-rays are generated from inside the oral cavity and the film is wrapped around the face. (Prasanna Kumar, Bailoor DN 2004. Yenepoya Dental College and Hospital, Mangalore)

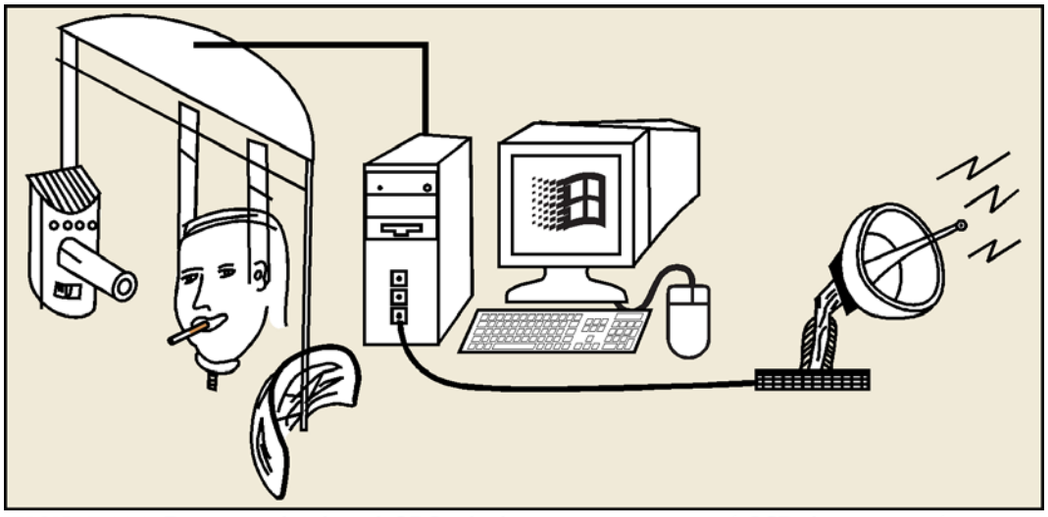


FIGURE 29.3: Depicting the digital OPG which contains an array of CCD detectors, software to process the image, hardware to show the image and internet to send the image anywhere in the world. In the next ten years the transition from film based imaging to digital film less imaging will be complete. (Prasanna Kumar, Bailoor DN 2004 Yenepoya Dental College and Hospital, Mangalore)

FIGURE 29.4: Showing OPG printed out from digital OPG machine (Courtesy: Umarji H, Ani John GDC Mumbai 2004)

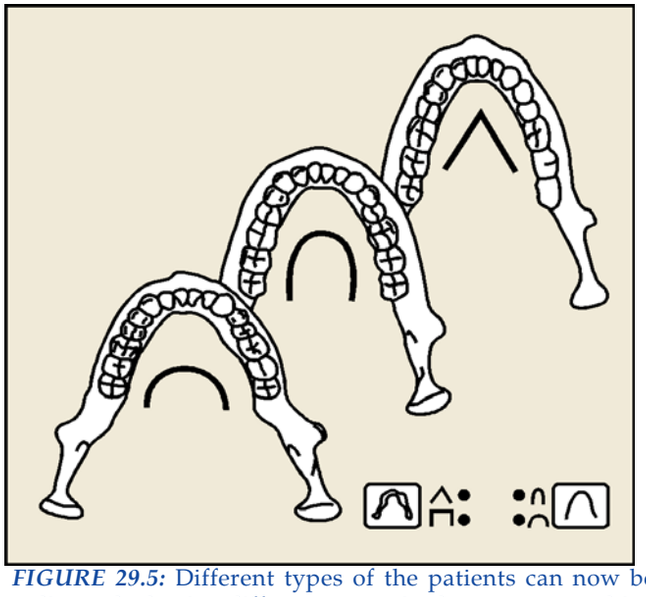


FIGURE 29.5: Different types of the patients can now be radiographed using different customized programs resulting in finer details in the OPG film (Prasanna K, Bailoor DN 2004) (Figure courtesy of Messrs Empire Marketing, Mumbai –for Planmeca OY, Finland 2004)

During the continuous exposure the rotation center is once again shifted so that movement pattern and, the projection of the jaw are symmetric on both sides[8] (Fig. 29.5).

- Continuously moving rotation center It is not necessary for rotation center of the beam to be stationary at any instant during the exposure and the beam may be given a sliding movement throughout the total excursion so that the effective projection center (functional focus) is continuously shifted along a defined path.
- Combined stationary and moving rotation centers Different combination of the principal movement described previously have been applied in practice.

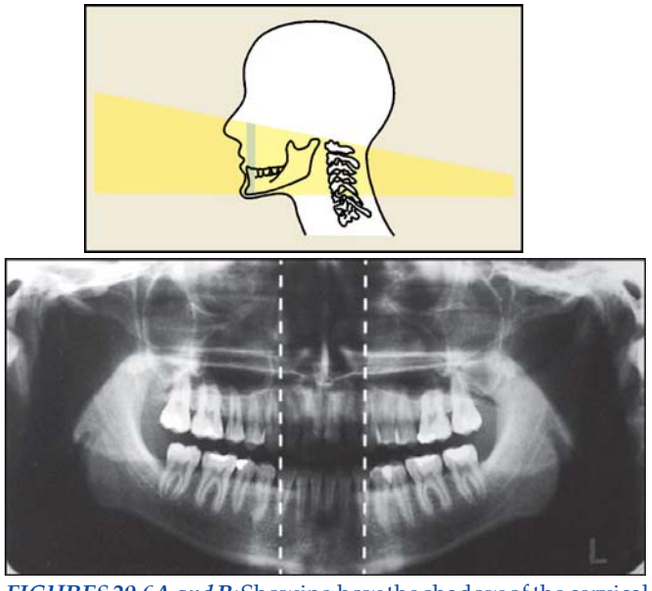
PATIENT POSITIONING AND ESTABLISHING EXPOSURE FACTORS

Patient Positioning

It is imperative that the patient be positioned correctly in

the panoramic machine, so that the jaws are within the focal trough.

1. Patient should remove the sweater or jacket so that there is space between bottom of the cassette holder and the patient's shoulder.
2. Patient should stand erect or straight with back straight.
3. The patient's head should be tilted downward so that the tragus-ala line is 5 degrees, down and forward. This will position the occlusal plane slightly downward towards the floor. In patients with high arched palate, decrease the occlusal plane angulation slightly and *vice versa*.
4. Align the mid-sagittal plane of the patient's head with the vertical central line of chin rest.
5. Explain the patient how the machine works.
6. Insert a cotton roll or a bite block between incisal edge of the maxillary and mandibular teeth.
7. Have patient close the lips and place the tongue against the roof of the mouth.
8. The patient should remove glasses, and metallic items from head and neck like necklaces, neck chain, napkins, nose jewellery, earrings, hearing aids, hairpins, zippers, etc.



FIGURES 29.6A and B: Showing how the shadow of the cervical spine can produce a discontinuous picture in the midline region. Some OPG machines alter the speed of rotation to compensate for the image density and contrast (Courtesy of Messrs Empire Marketing, Mumbai –for Planmeca OY, Finland 2004)

- Establishing exposure factors depending on the type of panoramic unit used and the film screen combination, appropriate kVp and or mA recommended by the manufacturers should be selected. The exposure time is fixed in the panoramic radiography.

Exposure factors (kVp and/or mA) should be adjusted accordingly when taking a panoramic radiograph under the following circumstances.

- If the patient has darker skin pigmentation, use higher kVp/mA setting.
- If the patient has heavy soft tissue and/or bone structure above the face and neck, use the next higher kVp or mA setting.
- If the patient is small and has narrow facial bone structure, use the next lower kVp and/or mA setting.
- If the patient is edentulous, use the next lower kVp and/or mA setting.
- Radiation dosage Tammisalo EH et al (1964)[11] reported that with the jaw orthopantomography, the patient received a total radiation dose of 4,600 mR when compared to the full mouth exposure dose of 12,800 mR.
- Intensifying screens, panoramic films and processing The phosphor originally used in X-ray intensifying screen was crystalline calcium tungstate (CaWO4). It is used in panoramic screens and is still the most widely used phosphor. It produces light in the blue region of visible light spectrum, and panoramic film is sensitive to most of the light emitted by this screen. Panoramic film is not sensitive to red light, so dark room safe lights with dark orange (Kodak 6Bx) and brown filters, (Kodak 6c) are recommended.

Some of the newer films, such as Kodak or G, a slow speed film used with Kodak lanex-rare earth screens are green light sensitive and require reddish brown filters (Kodak GS-1).

Several factors determine the speed of the intensifying screen including the thickness of the phosphor layer, the size of phosphor crystals, the presence or absence of light absorbing dyes, and the conversion efficiency of phosphor crystals.

High speed screens record less details. Since 1973 newer phosphors have been developed for intensifying screens including Barium fluorochloride, Barium strontium

sulphate, Yttrium oxy sulphate, and rare earth gadolinium and lanthanum.

There are various types of panoramic films available usually, there are 5″ × 12″ or 6″× 12″. Panoramic film speeds are rated slow, medium, and fast.[8]

FIGURE 29.7: Promax digital OPG manufactured by Planmeca OY, Finland is one the latest OPGs in this series. Many brands have come all over the world and you must check which company gives a good after sales service in your region (Picture kindly supplied by Messrs Empire Instrumentation Mumbai)

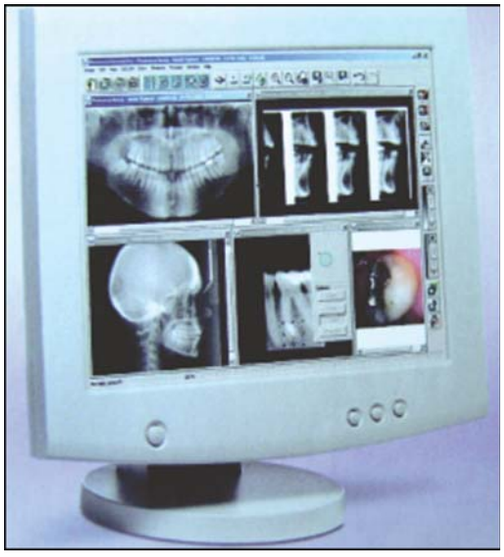


FIGURE 29.8: Showing the digital output from an OPG machine with latest CCD sensors. Trade name is Promax Digital Courtesy of Messrs Empire Marketing, Mumbai—for Planmeca OY, Finland 2004

Panoramic Ghosts

(Panoramic ghost otherwise known as reverse shadow, secondary image, inherent image, shadow reverse image, contralateral image.) (Fig. 29.6).

All objects or object details scanned by the beam in the rotational panoramic radiography will be projected on to the film.

It applies to all structures positioned on the side of the jaws, opposite the side within the layer, which is of primary interest. In certain instances, objects with a high attenuation may be observed in two positions in the panoramic radiography.

One of these images is real, and the other is usually referred to as ghost image. The ghost image is recognized by an un-sharpness, which in horizontal dimension is marked and is always projected at a higher position in the radiograph than its real counterpart. It is projected at a higher level because the beam is directed from below. Another important features of ghost images is that they are always reversed (Fig. 29.9).[6,9]

The characteristics of ghost images mentioned are:

1. In most cases it will be seen at a higher location than the primary object.
2. It will always be distorted, especially the horizontal component.
3. It may not be seen on a clinical radiograph if superimposed over area of dense anatomy.
4. It may appear even if the primary object is not seen on the radiograph.
5. It will always have some degree of radiopacity.
6. Pronounced horizontal blurring indicates that the primary object was at or close to a center of rotation.
7. It is reversed when compared to primary image.
8. It can be caused by physical objects such as earrings, a napkin, chain, a necklace, a zipper on a pull over sweater, an amalgam restoration or crowns and radon or radium implants.
9. It can be caused by anatomic structures, such as body and ramus of the mandible or cervical vertebrae.
10. It can be caused by parts of panoramic machine such as the chin rest or letters R and L on the head position.

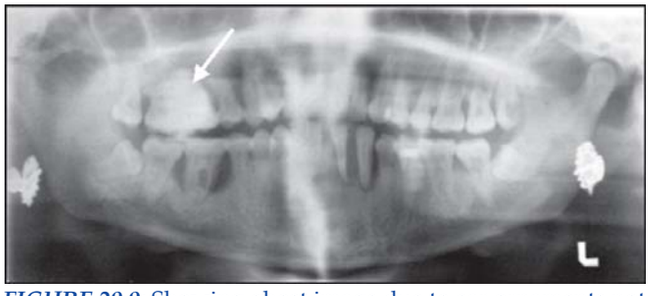


FIGURE 29.9: Showing ghost image due to ear ornaments not removed in the patient (Prasanna K, Nillofer S, Bailoor DN 2003 Yenepoya Dental College and Hospital, Manglore)

11. It can be pathologic. For example A sialolith or an impacted third molar. The characteristics of a double images are: (1) It is caused by mid line objects. (2) The bilateral images are of comparable clarity.
    - Faults in panoramic radiography to produce panoramic radiographs with constant image quality, common errors must be kept to a minimum. The errors can be (1) positioning errors. (2) Faults in film exposure, processing and handling the film.
- Faults in positioning the patient [11]

I. ***Occlusal plane positioning error***: If the occlusal plane is positioned parallel to the floor rather than slightly down and forward it produces, (a) A superimposition of the hard palate or maxillary tooth apices, (b) A loss of density in the middle of the radiograph, (c) The loss of either one or both temporomandibular joints, (d) when the chin is tipped upwards, it causes magnification of maxillary anterior teeth.

Downward overangulation (tragus-ala line greater than 5 degrees downward and forward results in a severe curvature in the occlusal plane and consequent lack of definition of the incisors on the radiographic image).

II. ***Distortion***: Patient movement during exposure could give a series of artifacts or distortion effects that are localized to only one region of the radiograph, namely the region where the rotating beam was scanning when the patient moved (See Fig. 29.14).

a. If the patient moves slowly in the same direction as the beam movement, the same detail will be exposed for a prolonged time. All details in that region will be stretched out in the image, the horizontal dimensions will be increased.
b. If the patient moves slowly in a direction opposite the beam movement, the horizontal dimensions of the image in the region in which this movement occurred would be decreased.
c. If the patient moves suddenly in the same direction as that of the beam movement, this region may be portrayed twice in the image.
d. If the patient makes a sudden movement in the direction opposite to the beam movement, a part of the object will be missing in the image.
e. If the patient moves upward or downward during the exposure, it causes an indentation in the lower border of the mandible, which may mimic a fracture.

III. ***Errors in positioning the cassette***:

a. Alternate vertical dark and light bands on the radiograph results from improper movement of either the cassette or the tube head-cassette holder assembly around the patient's head. This may be due to patient's shoulder touching the cassette holder during its movement in the exposure cycle.
b. When the cassette is placed too far below the mandible, the diagnostic information in the maxilla will be cut off.
c. When the cassette is placed backwards, metal lock and spring will be projected as radiopaque in the radiograph.

ADVANTAGES AND DISADVANTAGES

As with any new techniques OPG radiography has its advantages and disadvantages.

Advantages

1. **Size of the area radiographed**: The OPG covers an area that includes all of the mandible from condyle to condyle and maxillary region extending superiorly to the middle third of the orbits. Areas such as condyles, inferior border, angle and ascending ramus of the mandible, and entire maxillary sinus that are not visualized in intraoral surveys are seen routinely on OPG. Many diseased areas that might go undetected on intraoral surveys will be seen on OPG.[2-4]

2. **Simplicity:** OPG procedures are relatively simple to perform.[1,11]
3. **Patient co-operation:** Since OPG is an extraoral procedure, it requires a minimal amount of patient's co-operation. Patients are only required to be still for 15 to 22 seconds of exposure. Most units can be operated without radiation to demonstrate to the patient what the procedure will be like before the actual exposure will be made. It virtually eliminates problems with gaggers, patient with trismus, and fearful or uncooperative children.
4. **Time:** Less time is required to do an OPG examination than intraoral survey.[3]
5. **Dose:** Radiation dose to the patient is relatively low,[4,10,11] when compared with conventional full mouth intraoral radiography.
6. **Patient education**: OPG films are a valuable aid in patient education and case presentation. Conditions such as impactions, eruption patterns of teeth, the need for replacement of missing teeth, and fractures are more easily illustrated on panoramic views.[3]
7. **Discourage general screening OPG**: Many of the general practitioners may request for a general screen OPG. According to Rushton VE et al [20] 1999 only when specific indications like locating a deeply impacted wisdom tooth, initial look at TMJ or a lesion which spans at least half a jaw is suspected, a OPG may be safely advised considering the risk versus benefit factor.

Disadvantages

1. **Image quality**: Tomograms inherently show magnification, geometric distortion and poor definition. Because of poor definition panoramic radiography is less effective in detecting early interproximal or recurrent caries, disruptions in lamina dura, loss of crestal alveolar bone and thickened periodontal membrane.
2. **Overlap:** OPG units have a tendency to produce overlapping of teeth images, most particularly in the premolar area.[3]
3. **Superimposition:** There is often superimposition of the spinal column on the anterior portion of the OPG.[3]
4. **Distortion:** The amount of horizontal and vertical distortion varies from one part of the film to another. This results in an uneven magnification of the image and structures and spaces may be seen larger than actual size.[8]
5. **Overuse:** The ease and convenience in obtaining the OPG might lead to carelessness by substitution for other projection that might be adequate. This is one of the prime concerns in regard to patient dosage.
6. **Cost:** Because of its high cost it is an extra investment for practitioners.

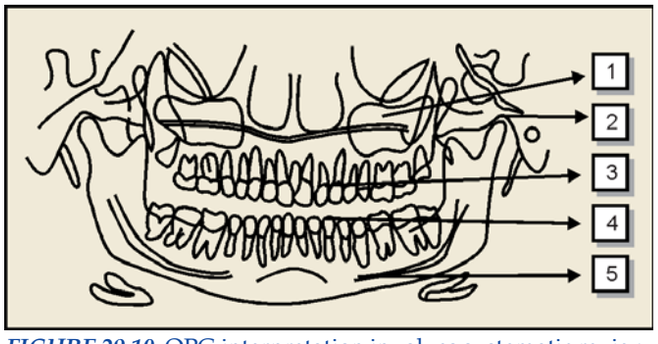


FIGURE 29.10: OPG interpretation involves systematic review of the film. Even if obvious pathology is evident continue your systematic sweep over the film we suggest a five level slow sweep for optimal interpretation—1. Shows the maxillary sinus, 2. TMJ regions, 3. Maxillary occlusal sweep of the eyes, 4. Mandibular sweep of eyes, 5. Lower border of mandible and include the styloid process and hyoid in the interpretation. (Prasanna Kumar, Bailoor DN 2004 Yenepoya Dental College and Hospital, Mangalore)

Interpretation[13]

The problem in interpreting panoramic radiograph is the manner in which the panoramic projection "Spreads out" the structure being reviewed (see Fig. 29.10). The panoramic radiograph is unique in that the foci of projection in the vertical and horizontal direction are not the same. In horizontal direction it is the rotational center of the beam that constitutes the functional focus, where as in the vertical dimension it is X-ray source. In rotational panoramic radiography the film is not stationary, but moving this affects the length of the image recorded in the film, thus the moving film changes the horizontal magnification of the image but not the vertical projection of the object. Thus the horizontal measurement in the panoramic radiography is not reliable. Also because of poor definition, they are less effective in detecting early interproximal or recurrent caries, disruption in lamina dura and loss of crestal alveolar bone.[8]

To accurately interpret the OPG images it is imperative to assimilate the following concepts.

- *Interpretation pointer 1*—The structures seen are actually flattened out; it is as if the spine has been vertically split and each half folded up outwards with TMJ, sinuses and the mandible.
- *Interpretation pointer 2*—The real image is formed when the object is close to the focal trough, and sometimes the midline structures may be projected as single or double images. The structures that occur in a diamond shaped region where the two axes of rotation meet occur as double, thus positioning errors in the horizontal planes can make the turbinates and meati to occur double in the sinus region or the hyoid to occur double.
- *Interpretation pointer 3*—Ghost images are formed: A ghost image is formed when the object is located between the X-ray source and the center of rotation. The most common example is when the earrings of the patient are inadvertently overlooked and radio graphed, there would be four sets of images, two real and two ghost.

  Anatomic structures often ghosted are the hyoid bone, the cervical spine, and inferior border of the mandible.
- *Interpretation pointer 4*—Panoramic radiography beams are affected by some soft tissues to a sufficient degree to become visible in the radiograph, dorsum of the tongue and the postpharyngeal wall are the common examples.
- *Interpretation pointer 5*—Air spaces are visualized—Nasopharynx, maxillary sinus, and nasal fossa. Occasionally an air space is seen above the dorsum of the tongue and represents an error in technique, which can be avoided by asking the patient to place his tongue against the palate while the radiograph is being taken.
- *Interpretation pointer 6*—Separation of radiolucencies of patient and the machine is important. The objective of the whole OPG technique lies in the fact that single real image of the three components of the patients tissue, i.e. hard tissue, soft tissue and the air spaces is obtained.
- *Interpretation pointer 7*—Extradental coverage of the OPG is excellent. The areas viewed specially are TMJ, Styloid process, Paranasal sinuses, Sialoliths, Calcified lymph nodes.
- *Interpretation pointer 8*—The radiograph can be made in trismus cases, trauma cases, etc as a basic scout film. This will enable us to decide on further specific radiographs required.

The work of Chiles JL and Gores RJ[14] and that of Higashi T and Iguchi M[15] is specially recommended for the serious student of anatomic landmarks in OPG:

Akarsian ZZ [21] has evaluated the following errors in his study and found that ten commonest errors are

1. The chin of patient tipped too low—Occlusal plane sloping downward and chin placed upwards resulting in the occulsal plane sloping superiorly.
2. Bite block not placed or misplaced by patient—Overlap of the anterior teeth.
3. Midsagittal plane of patient and machine not coinciding—Asymmetrical magnification of teeth due to the improper rotation of the head. Ramus appears to be asymmetrically magnified .
4. Bite block too forward—Superimpostion of the spine on the condyles or rami or blurring of anterior teeth.
5. Bite-block too far back—Widening of anterior teeth due to the patient biting.
6. Non-removal of ornaments—Radiopaque artifacts (earrings, necklace, prothesis, lead apron, spectacles, etc).
7. Shadow above the tongue due to the patient not raising the tongue against the palate.
8. Vertebral column causing extreme lightness in the anterior region as a result of the superimposed shadow of the spine.
9. Dirty or dusty radiographs with folds causing artifacts
10. Lightning tree appearance is because of static due to friction between the radiographs while removing from packing or repacking unexposed films.

CONCLUSION

OPG view has become more or less a standard view when there is any trauma, large swelling or suspected tumor in any part of the upper and lower jaws. ENT and general physicians are increasingly looking towards this technique as a quick review technique. The midline shadows in the

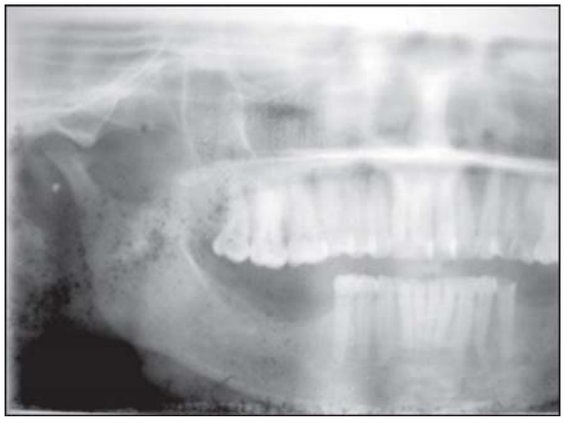


FIGURE 29.11: Showing inadvertent exposure to light in stored films—illusion of fracture is created due to the radiolucent region in the ascending ramus region. (Prasanna K, Nillofer S, Bailoor DN 2003 Yenepoya Dental College and Hospital, Manglore)

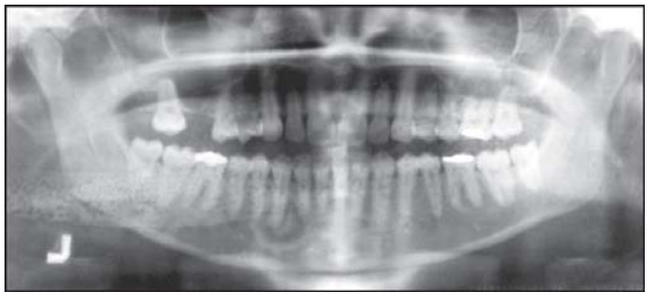


FIGURE 29.12: OPG showing the artifact caused by improper washing of the film resulting in the yellowish discoloration of the sulphur within a year of storage (Nillofer S, Prasanna K, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

FIGURE 29.13: OPG showing flash artifact in the picture. When the X-ray is to be copied on to a digital camera then switch the flash off and increase illumination from behind (Bailoor DN, Prasanna Kumar 2004 Yenepoya Dental College, Mangalore)

FIGURE 29.14: Showing improper vertical positioning of the patient resulting in the cutting off of the left side of the TMJ and ascending ramus. With recent machines laser cross hairs will disallow such errors (Bailoor DN, Bailoor Sonali 2004, Yenepoya Dental College and Hospital, Mangalore)

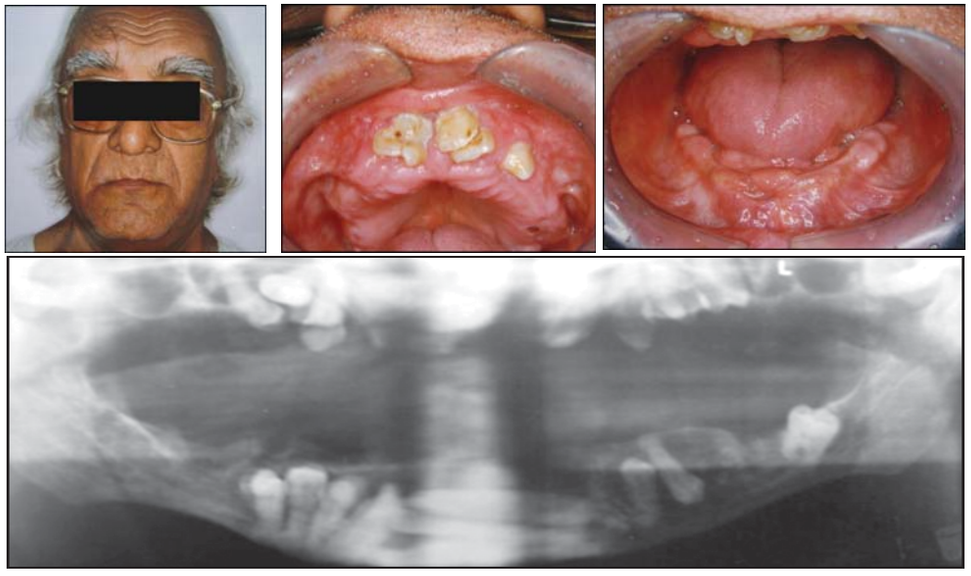


FIGURE 29.15: Showing the importance of OPG examination prior to complete denture prosthesis insertion. 60-year-old male who was wearing dentures for a period of 9 years suddenly developed illfitting dentures and loosening. Clinically teeth appeared to be erupting in the maxillary segment. OPG reveiled multiple unerrupted teeth (Courtesy Umarji H, Ani John GDC Mumbai 2004)

mandible are still not very sensitively visualized since the spine shadow still poses a definite irritation. The space between tongue and the palate also can be a distraction to a new clinician. Keeping in mind the limitations of this curved surface tomographic view it is possible to utilize the knowledge obtained from rationally for treatment planning (Figs 29.11 to 29.15).

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Bailoor DN

30 Computers in Dental Practice

INTRODUCTION

Computers have become a routinely used tool in health care and dentistry is not far behind. In fact use of computerized history formats, film less imaging and teaching programs using digital media have reached mundane levels of utilization.

With a strong broad band connectivity being taken up by private companies Internet has become a major platform for dissemination of information and e-commerce reaching the smallest villages in India.

Telemedicine, Tele-dentistry, and health diagnosis being given from remote locations by specialist doctors located in tertiary level hospitals has become a routine which Indian health care system needed very badly.

In the following chapter we shall discuss basic principles keeping well in mind that a lot of information given is likely to become obsolete in less than a year!

WHAT IS A COMPUTER?

A computer is an electromechanical device, which can process data and information to help in decision-making. This device is divided into two parts:

- Software
- Hardware

For the sake of understanding hardware can be compared to a human body and the software to the human mind. It is the software, which runs all the hardware as it has all the instructions for the hardware to behave and perform.

The hardware consists of: all the microchips, the central processing unit (CPU), visual display unit (VDU), mouse, keyboard, scanner, printer, digital camera, etc.

The software is like music on a magnetic tape. This is classified into 3 groups,

- Operating software: Unix, Linux, Windows 98, Windows 2000, Windows XP professional, Apple Mackintosh, OS2.
- Application software: Microsoft Word, Microsoft PowerPoint, Excel Tally, Microsoft Access, etc.
- Cystomised software: Visual basic or VB6. Doctor Master

The *operating software* is the basic software, which runs the computer when switched on. The *application software* is the one, which helps us to do day-to-day work.

The *customized software* is the one, which is designed to serve the particular needs of an individual such as a doctor or a clinic to present information in a particular format.

SPECIFICATIONS IN A COMPUTER

When acquiring a computer the technical details that need to be kept in mind are:

- Processor- Pentium 4 with a speed of 2.8 GHz or AMD Athlon XP.
- Motherboard.
- A hard disc drive (HDD) of capacities 40 GB or 80 GB.
- CD-ROM drives with CD-RW, CD-writer (52×24×52).
- Additional 1.44 MB Floppy Disc Drive (FDD), which is now almost obsolete.
- An uninterrupted power supply (UPS) unit with a spike buster is a must in Indian conditions to protect the device from fluctuations in the power supply.
- Proper earthing of the electrical circuit is an absolute must for safety of the device and it's operator.
- The multimedia kit including the keyboard, speakers, media player, etc are useful for making effective presentations, etc.

When a computer is purchased, also keep in mind the fact that a reputed firm must be given the responsibility of maintenance of the computer. Also the information in the device needs to be kept away from the prying eyes of unauthorized individuals, so safety devices such as digital locks, password guarded files, etc must be used.

COMPUTERS IN A DENTAL CLINIC SET-UP

In the bygone millennium computers were a luxury afforded by a selected few who displayed it proudly on their mantle pieces as a symbol of their affluence. The situation today has changed so much that even in remote villages, computers are a common thing.

The contemporary dental clinician needs to possess a computer for efficient functioning and optimum work satisfaction. The dental clinic may be a single computer clinic, where the clinician keys in the case history, show patient his pictures of treatment to be accorded and his previous radiographs.

When the clinic is a two-computer clinic, the receptionist can enter basic data in the patient's file, treatment and payment details can share a part of the duties. This type of set-up is ideal because this saves Chairside time of the dentist (Fig. 30.1).

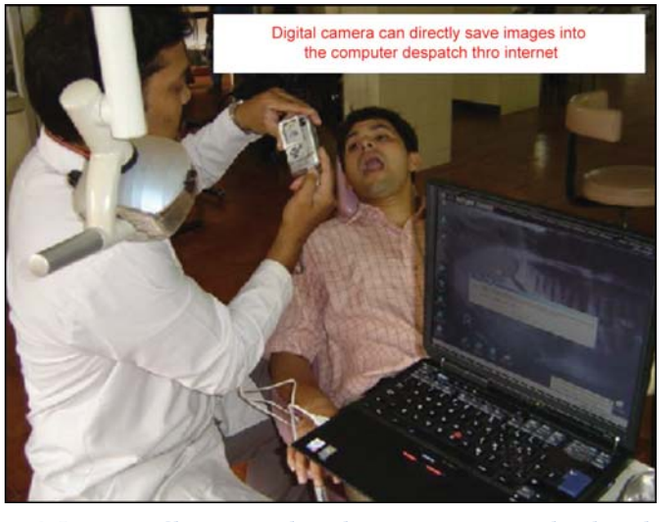

FIGURE 30.1: Showing a digital camera in use in the dental clinic. Images of lesions and can be saved, retrieved and sent over internet (Bailoor DN, Sahm K, Prasanna K. Yenepoya Dental College and Hospital, Mangalore)

COMPUTERS IN HOSPITAL SET-UP

In the hospital the computers become a tool for patient care and administration. Patient care and financial matters get inter twined so we usually need an integrated package, which will take care of all the aspects of hospital and teaching management. Many such packages have been designed in India and we just give a glimpse of the possibilities in following paragraphs.

COMPUTERS IN A DENTAL HOSPITAL

Computers are a basic necessity in teaching institutions. In the current scenario, most of the dental hospitals are making use of computers for teaching and administration. A dental hospital with routine functioning needs a Local Area Network (LAN) of one or more servers. Normally it is a good idea to have two functional areas of administration and patient care/research into two separate servers. These servers are in turn connected to a series of computers known as "*thin clients*" which use the client server type of architecture (Figs 30.2 and 30.3).

EXPERT SYSTEMS

The types of computer systems on the basis of their functional properties are:

1. Computational
2. Data processing
3. Knowledge processing

Table 30.1: Different expert systems used in hospitals

S. no.	*Name of software*	*Company*	*Address*	*Domains covered*
1	Q1HMS	QUAD ONE TECHNOLOGIES	#306 Flora Apts, Rd no 3, Banjara Hills, Hyderabad –34 Ph: 040-23350221 WEB www.quadone.com	Admin, OPD, In patient, Ward Management, Billing, Pharmacy, Stores, OT, Casualty, Medical Records, Blood Bank, Account/Marketing
2.	CIFT Health V 3.0	Ciftech Solutions Pvt Ltd	#71/1, 2 floor, 17 cross, Margosa Road, Malleswaram, Bangalore-55, Ph: 080- 3345123 WEB www.ciftech.net	Billing, OT, cash and accounts, stores, HRD, Patient ID
3	Health Space	Accel ICIM Systems and Services Ltd	#75, Nelson Manickam Road, Chennai-29 Ph: 044-23741856 WEB www.accelicim.com	Patient care, administration and resource management, finance management Clinical Data management, Diagnostic and Therapeutic Services, Materials and Asset management
4	MLL-Hospital	Microtechnologies (India) Ltd	Ph: 022-27878324 WEB www.microtechnologies.net	Alerts and Emergency Messaging, Reminders, Greetings, Group Messaging, Privacy and Security, Patient Care, Hospital Management
5	MediTrack	TechTrek Technologies Ltd	Plot no 124, Road no 17, MIDC, Andheri (E), Mumbai-93. Ph: 022-56936446 WEB www.techtrek.co.in	Back office, OPD, IPD, OT, Medical Records, Laboratory, Inventory, Accounting, Pharmacy, Blood Bank, Nursing Management, Biomed Engineering, Maintenance, Waste Management, Laundry, Rehab Management
6	PLUS SOFT HMS	Iden Techsoft Pvt. Ltd	#106/1, Kodandrama Complex, Gandhi Bazar Main Road, Basavangudi, Bangalore- 04 Ph: 080- 51204466 WEB www.iden-group.com	Patient registration, OPD, OT, Billing, Stores, Pharmacy, Accounts, Payroll, Records, Housekeeping, Ambulance Management
7	WIZCARE	Wintex Infotech Solutions	Miranda complex, Bondel, Mangalore-04 Ph: 0824- 2485315	Reception Query System, Patient Billing, Records, Pharmacy, Lab Management, Ward Management, Inventory, Human Resource Management
8	EasyHMS	Infolife Technologies Pvt Ltd	#334/1420, 22nd Cross, 1st $3rd Block East, Jayanagar, Bangalore-11 Ph:080-6341386 WEB www.infolifetech.com	Registration, OPD, IPD, Insurance Management, Records, Billing, Casualty, Laboratory, Store and Inventory, Pharmacy, OT, Housekeeping, Digital Signing, Reports, Diet Management.

Those systems that use computational information technology to calculate large complex equations based on pixel and voxel studies. Such systems are made use of in imaging machines like CT scanner, MRI scanner, and Radiovisiography scanner, etc these type of systems are known as *Computational systems*.

Different types of description of diseases, including classifications such as ICD-10 with various epidemiological variables and patient data can be interpreted. These can then be printed as reports. From these reports different levels of decisions can be taken. Incidence and prevalence of diseases can be known using these systems. Such systems used to process large quantities of data are known as *Data Processing Systems.*

The refinement of data processing is taken to the next level in *Knowledge Processing Systems*. Here rules are instructed to the software and data when put in, is analyzed and the report of significance flashes onscreen. This is

then used in decision-making, wherein decisions are based on sound standardized patterns obtained from the system.

The systems described above are called as *Expert Systems.* An *expert system* is a computer program intended to embody the knowledge and ability of an expert in a certain domain.[1] These are used in various industries, such as in health sciences for diagnosis and treatment (Table 30.1), in financial planning by stock advisors, automated control of manufacturing units, auto pilot functioning in air services, determination of physical and chemical properties of unknown compounds, in the lung-heart machines used in cardiothoracic surgeries to name a few.

- The expert systems have an explicit knowledge base.
- They use symbolic logic rather than just numerical calculations.
- These have the ability to explain its conclusions with ideas that are meaningful to the end user-the clinician.

The expert systems have the advantage of being consistent with comprehensiveness and ready availability. In earlier computations, only YES or NO logic was being applied. However intelligent choices sometimes lie in the realm of uncertainty. To face all these types of uncertainties and fuzziness, expert systems use the following methods:

- Bayesian probability
- Fuzzy logic
- Belief networks
- EXSYS approach

Medical expert have been mainly used for:

- ***Generating alerts and reminders***: in real clinical situations, heartbeat, ECG readings and laboratory values can be monitored and whenever they exceed normal value suitable alerts are generated.
- ***Diagnostic assistance***: when rare cases are discussed and classified latest information can be downloaded and an ICD type of classification done instantly.
- ***Therapy planning***: expert systems are used to look for inconsistencies and omissions in an existing treatment plan, and use multidisciplinary input for better results.
- ***Education*****:** expert systems are used to train and allow clinicians and students to practice various medical tasks.

The medical expert systems that are commonly used are:

TxDENT: It is an expert dental diagnostic screening and tracking system.

Jeremiah: It was designed to provide dentists with orthodontic treatment plans for cases suitable for treatment by general dental practitioners with knowledge of removable orthodontic techniques.

Orthoplanner: It is a knowledge based system to provide dentists with orthodontic treatment plans for cases where fixed orthodontic appliance techniques must be employed.

RaPiD (Computer Aided Partial Denture design): RaPiD is a knowledge-based system for designing Removable Partial Dentures (RPD).

SETH: It is an expert system for the management on acute drug poisoning.

MYCIN: It is a program for advising physicians on treating bacterial infections of the blood and meningitis.

USES OF COMPUTERS IN A GENERAL DENTAL CLINIC

Record Keeping, Accounts and Administration

- These are a fixed feature of any organization.
- They build and maintain patient database. Detailed patient history with allergy profiles, etc can be maintained.
- All the fee details (paid and in balance) and account books can be kept up to date.
- Appointment scheduling of patients can be done conveniently without the risk of overlaps.
- The computer can generate reminders for appointments, birthdays and other such alerts.
- Stock of medicines and materials can be monthly reviewed, if daily usage data are entered.
- All the addresses of professional colleagues, patients, etc with their contact phone numbers and e-mail ids can be stored electronically.
- The payroll of all the employees in the clinic can be maintained conveniently.

Practice Management

In the modern society, a professional approach to managing a clinic is warranted. Such management

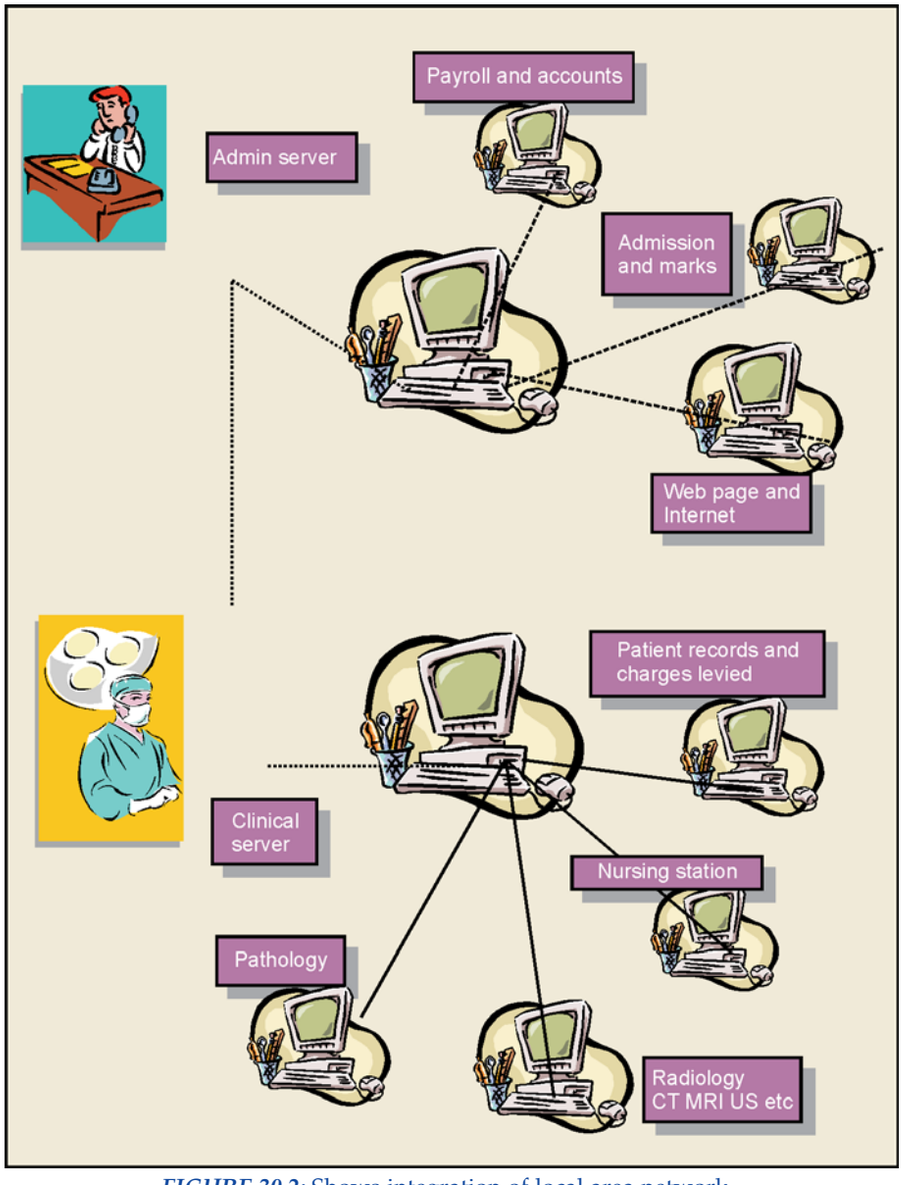

FIGURE 30.2: Shows integration of local area network

softwares are available in the market, which help in maintaining data related to the day-to-day functioning of the clinic. Any lapses in the productivity on the part of the dental team staff versus the cost of functioning can be analyzed using these programs.

Patient Education

In India awareness about oral conditions is scarce. Patients are unaware of routine procedures done in a dental clinic and are hence very apprehensive of attending such a clinic. Effective software programs are available which show all the details of the procedure to be performed in pictures and text, that the layperson understands easily. This is a boon to the busy clinician whose chairside time can be saved as the receptionist or auxillary can counsel the patient.

The clinical pictures of previously treated similar cases can be shown to patients for gaining a perspective into the anticipated results. All the treatment related pictures of a particular patient could be conveniently stored and retrieved at the click of a button.

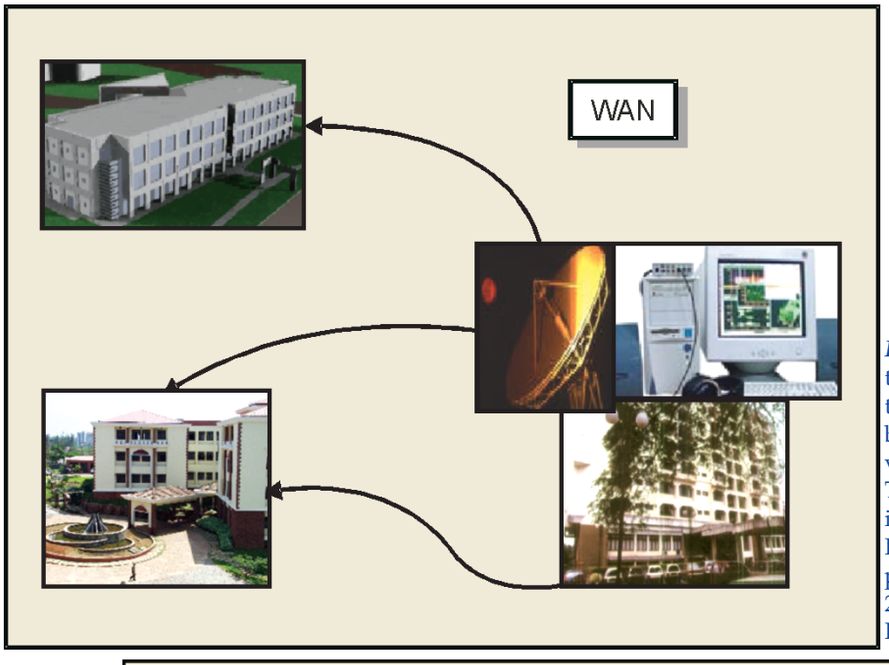

FIGURE 30.3: Wide Area Networks are used to connect hospitals in different parts of the town, and in different towns and use basically either extra terristerial connectivity via radiofrequency or via satellite networks. Terristerial cables of fiberoptic are also used in short distances. Guard software called as Firewall are used to maintain security and protection for the medical data. (Bailoor DN 2004, Yenepoya Dental College and Hospital, Mangalore)

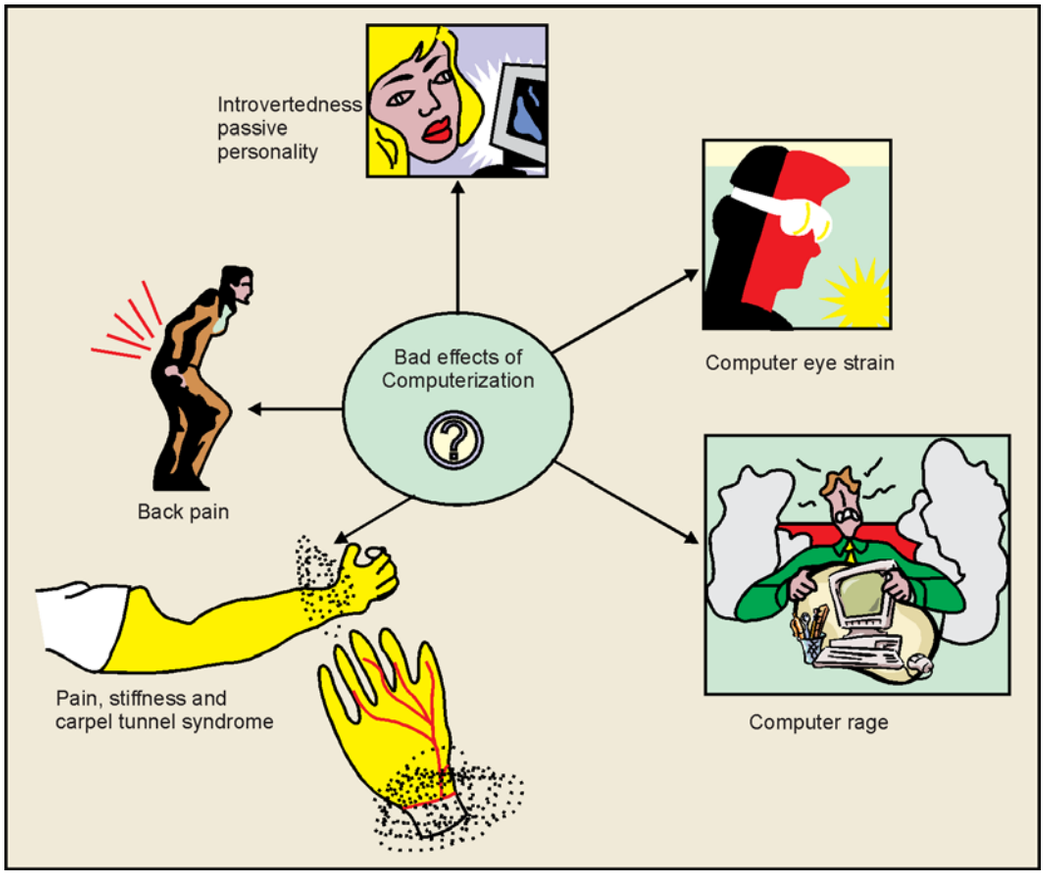

FIGURE 30.4: Rampant computerization has some side effects (Bailoor DN, Prasanna K, Nillofer S 2004, Yenepoya Dental College and Hospital, Mangalore)

VIRTUAL REALITY (VR)

Virtual reality (VR) is gaining recognition for its enormous educational potential. While not yet in the mainstream of academic medical training, many prototype and first-generation VR applications are emerging, with target audiences ranging from first and second year medical students to residents in advanced clinical training. Visualization tools that take advantage of VR technologies are being designed to provide engaging and intuitive environments for learning visually and spatially complex topics such as human anatomy, biochemistry, and molecular biology. These applications present dynamic, three-dimensional views of structures and their spatial relationships, enabling users to move beyond "real-world" experiences by interacting with or altering virtual objects in ways that would otherwise be difficult or impossible.[4]

The educational effectiveness of VR didactics has been limited to empirical pilot studies. Tests examining the VR learning experience of children showed mostly positive results. Adult learners have not that frequently been included in evaluation procedures so the differences between adult and child VR teaching methods still remain unclear. New visualization tools, such as endoscopy, are able to broaden the student's medical outlook and strongly reduce the dependency of dissection. The VR training and learning system can be used both locally and remotely, in a distributed manner on a global level.

Virtual reality technology is rapidly moving into the operating room and the medical school. With phantoms, surgeons are practicing new endoscopic techniques by watching their movements on a computer screen or through virtual reality goggles-and feeling the tension of moving through human tissue-as they manipulate surgical tools in life-like situations. Mannequins are reproducing in exquisite detail as many as 80 different physiological events in reaction to the actions of surgical residents. Full-scale simulated operating rooms, complete with working monitors, are recreating surgical crises for teams of faculty and surgeons-in-training.[3]

USE OF COMPUTERS IN DIFFERENT SPECIALITIES OF DENTISTRY

A representative list of computer usage in the different departments of a dental school is mentioned below. It is by no means complete, since with each passing day we are constantly witnessing the evolving advances in computer technology with newer implications for educational, research and therapeutic purposes.

Oral medicine and radiology:

- Patient history recording in a local network or online. Patient need not carry records to each department as all the details can be viewed electronically.
- Identification of medical risk.
- Physician referral letters and written statements for treatment of medically compromised patients.
- Allergy profiles and links to the prescription generators.
- Archiving system—records, photographs, radiographs, treatment details, fingerprints (for forensic interest), etc can be stored.
- Dental imaging using intraoral camera connected to and controlled by computer can be installed in the dental chair unit. It can be an effective record keeping, educating and marketing tool.
- Radiovisiography—radiographs stored in the electronic form, by digitizing it can be used for radiographic analysis of landmarks and radiographic lines and planes.
- The panoramic radiographic unit, an essential requisite, is totally computer controlled.
- ORASCAN and OralCDx which use a brush biopsy/Exfoliative cytology procedure and subsequently use computerized image processing to give a standardized report are being utilized in some advanced countries like USA but yet to be commercially available in India.[2]

Oral Pathology

- Three-dimensional imaging with visualization of blood vessels has helped in histopathology of tumors. A special stain called PECAM-1 seems to be very useful.

Hama Y et al of the Shinshu University School of Medicine, Japan have used a special 3D structure volumetric study after staining with PECAM-1 with ABC technique (the avidin-biotin complex). This technique uses computerized image analyzer which gives a detailed structure tree of the micro-blood vessels.

- Rashbass J has mentioned how many laboratories use the coding systems such as SNOMED for storage and retrieval of histopathology slides. It is possible to store histopathologic images in the hard disc of the computer using a digital camera mounted on the high power microscope. The images can be very easily sent via e-mail or printed out for reference.
- Other specialists in the hospital can access digital histopathologic images from the pathology laboratories over LAN.

Periodontia

- Standardization of pocket depth measurement by use of computerized probes using softwares specially created for the same purpose.
- Measurement of alveolar crest levels using digital subtraction radiography.
- Occlusal stress management by using sensors on selected areas of teeth suspected to be under abnormal stresses using feedback from a computerized system.
- Electro-myographic monitors with special computer programs to check for muscle imbalances and traumatic occlusion are used.

Conservative Dentistry and Endodontics

- Computer aided Caries Diagnosis- Newer softwares are being developed which combine the radiovisiography with some form of an assisted decision making systems, in order to diagnose caries. Ganbegovic et al.
- Radiovisiographic unit for on the spot endodontic decisions.
- Computerized apex locators and pulp testers attached to PC with different diagnostic programs.
- Use of image modification programs for showing impact of cosmetic dentistry on patient's appearance by using simulation modes even before doing the treatment.

Oral Surgery

- Keeping track of the postoperative complications.
- Orthognathic surgery planning and mock surgery can be performed using special programs.
- Virtual reality training programs can help train reflexes of new surgeons by making them do a surgical procedure in VR environment in which any number of incisions and suturing can be practiced.
- Use of computer aided design and manufacture for generating 3D models in balsam wood or plastic, again for purpose of mock surgery.

Orthodontia

- Computerized cephalometric studies are routinely being done today in major cities in India. Orthodontists are using indigenous softwares designed by AIIMS, New Delhi based on Indian normal values (DIGICEF).
- Orthognathic surgery, digitized laser holograms are being used to do a detailed analysis of soft tissue profiles in assessing their role in aesthetic imbalance of the patient.
- Research on various aspects of malocclusions by analyzing the epidemiological data using statistical packages (SPSS®) in the selected population of the area.

Prosthodontics

- Onscreen measurement of digitized radiographs prior to implant placement.
- Prosthesis being manufactured by the CAD-CAM (computer aided design- computer aided manufacture) type of devices in which digital measurements of dental structures are made and the prosthesis is designed onscreen which is milled out of a solid block of metal. This can be used for direct cementation on a broken down tooth.

Community Dentistry

- Detailed planning of dental camps and generating statistics can be done.
- In depth study of epidemiological data on treatment needs and availability of dental services to advise government on the areas to be developed for general and speciality services in India.

- Studies of relevant patterns of social factors, sexual habits and traditions in spread of insidious diseases like AIDS, etc.

In addition the computer systems can find use in the library for reference retrievals, translation of foreign works, translation of english work into local languages for distributing into the village levels by special software and for publishing of in-house journals and local research findings for immediate distribution and teaching. Routine administrative work can be done efficiently and accurately using LAN. No professional library is complete without a computer system.

INTERNET AND RELATED ISSUES

The Internet is a virtual world wide web (www) network. It can instantly connect to any part of the world via satellite uplink. It has opened a plethora of choices for all of us. It brings an enormous quantity of information and data at our fingertips. Efficient usage can transform any level of activity and dentistry is not an exception. Today even the remote towns and villages have been penetrated by the cyber cafes. The net can be easily and inexpensively accessed there. The general public can immensely benefit if taught the correct use of the net.

However there exist some limitations in its use!

- The net is not designed for efficient and specific information retrieval. Many search engines are available, but they are not consistent and sensitive.
- Many difficulties are encountered at some websites.
- When some information is found, the accuracy and validity of such matter cannot be ascertained.
- Also the information posted on the net can be manipulated through website hacking.

USE OF INTERNET IN DENTISTRY

- The Internet can be an efficient marketing tool for practicing clinicians. The dentist can effectively showcase his practice philosophy, quality of care and familiarize the potential patient in a creative manner by hosting dental practice websites. However the limitation of this opportunity is that only those who surf the web can get acquainted with a dentist in this manner.
- Patients can be reminded of their appointment schedules via e-mail.
- All the latest advances being made in different parts of the world can be accessed easily. The websites displaying current trends in research can be logged on to easily.
- The concept of tele-dentistry is fast catching up. In remote villages where sufficient dental care services are not available, the specialist in a tertiary care center can be consulted via the Internet. The opinions of other consultants can be obtained by e-mailing them the clinical pictures and radiographs. Discussions among various specialists can be easily made without all of them having to be present at the same place.
- Tele-dentistry can be practiced in two ways
- Using the file transfer protocol, where the files of data are stored and then forwarded via the net.
- Live video conferencing and data conferencing is the other mode.
- Continuing dental programs can be conducted via the net, where clinicians in different parts of the world can easily access the information.

Dental Resources on the Internet

1. American dental association- www.ada.org
2. Bad breath research—www.tau.ac.il/~melros/welcome
3. British dental Journal—www.bdj.co.uk
4. Clinical research associates—www.cranews.com
5. Dental bytes magazine—www.sybor.com/dental-bytes
6. Pubmed—www.ncbi.nlm.nih.gov/pubmed
7. Journal of contemporary dental practice www.thejcdp.com
8. Postgraduate medicine journal www.postgradmed.com
9. eMedicne—www.eMedicne.com
10. British medical journal—www.bmj.co.uk
11. Free journals online—www.freejournals4doctors.com
12. Free textbooks online—www.freebooks4doctors.com
13. TMJ Tutorial—www.rad.washington.edu/Anatomy/TMJ

The general search engines that search for any matter on the Internet are available. These are:

- www.google.com
- www.alltheweb.com
- www.altavista.com
- www.rediff.com
- www.yahoosearch.com
- www.msnsearch.com

ADVANCES IN COMPUTERS

When the first computers were used they were bigger than rooms and were so huge that to repair one it took days. Now the computer has metamorphosed into a compact form as the desktop, which is very common usage. However the human quest to manufacture everything in miniature models has also affected computers. The desktop went mobile by becoming a laptop. Sleeker models of these machines are now available with flat screens, which can be carried in a briefcase. However not content with it we now have notebooks.

From these now the concept of *hand held computing* has emerged. The hand held computers are known as 'Palmtops' or 'Pocket PC'. These conveniently fit into the palm and can be carried around in the pocket. They can be connected to the Internet via the mobile phones. This is important in emergency situations like drug overdose or allergy reported by a patient when the clinician is away from the clinic and is contacted over phone. He can then immediately connect himself to the drug databases and render effective solutions.

The hand held computer or palmtop is made up of following parts-

- Power button
- Microphone
- Screen
- Writing area
- Quick access buttons
- Jog wheel
- Stylus
- Battery
- Internal RAM/ROM memory
- Processor

The operating softwares in theses PCs are- the Palm OS, the Pocket PC and the Psion.[5]

DARK SIDE OF COMPUTERIZATION

Like any other technological change, computerization has its PROS and CONS. Bergeron BP commented on the dark side of the digital evolution and complained that the proponents of IT Usage do not warn about "carpal tunnel syndrome"- pain and stiffness in the wrist due to prolonged keyboard activity; strained eyes; sociological problems due to internet where introvertedness becomes accentuated and over dependence on computer systems can lead to total confusion when they fail. A lot of otherwise creative people are computer phobic and do not like to work with computers. In many telephone systems computerized voice seems to greet the caller in an impersonal way. In healthcare human-human interface is very important and the machine cannot duplicate the empathy and caring (Fig. 30.4).

CONCLUSION

With internet and cyber media becoming a day-to-day reality, today's dentist will have to rely heavily on his computer to update his knowledge, use internet based medical records systems to store data and send the patient pictures, radiographs and other information to specialist doctors for the purposes of tele-consulatation. On line CDE programs and rating of the knowledge by on line questionnaires will become a necessary for keeping a dentists registration valid. Already we see that IT enabled dentistry is the way of the present and the future.

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Bailoor DN, Mody RN

31 Forensic Odontology in Dental Practice

INTRODUCTION

Forensic odontology is the utilization of scientific dental knowledge for the furtherance of justice. This includes correct collection of dental evidence, proper documentation, awareness of the legal procedures, the presentation of the evidence in court of law and finally giving the accurate scientific interpretation of this evidence so that it may either confirm or deny the charges that are brought about on the accused.

The practice of this branch means having good contacts with the local police department, knowing the legal formalities of the country where you intend to practice and having full time attachment to a teaching medical college so that the facilities of the forensic medicine department may be utilized and dentist can take part in the autopsies with his forensic specialist colleagues. Lastly, having specific training in this branch helps; right now it is not available in India.

The dental tissues, the teeth, jaws and the parts of upper face skeleton are all relatively indestructible parts of anatomy. Fire, explosion, and dead body immersed in water for prolonged periods looses the skin and muscle visual identification becomes virtually impossible. In such cases the dental evaluation can throw very critical light on the age, sex and even if the person was a national or a foreigner.

Acton C et al[8] 1999 in their study from Queens land, Australia analyzed one hundred and six thermal injury deaths in children found that 73 percent died in house fires and that forensic odontology played a pivotal role in identifying them and establishing their ages.

STEPS IN COLLECTION OF EVIDENCE (Fig. 31.1)

a. **Approaching the scene:** It's a good idea to coordinate with the police pathologist and go to the scene together. Go around the perimeter and observe the body or bodies and make detailed notes. Polaroid or regular photographs are a matter of police requirement.
b. **Protective clothing and the ECK**: Evidence collecting kit needs to be planned well ahead. It must have sterile gowns, different types of examining instruments, spatulas, injections, forceps, surgical blades and handles of 11 and 15 numbers. In fact the dentist must make a suitcase with placements areas for different instruments that will foster mobility.
c. **A forensic van with all the equipment** including a mobile X-ray unit and a computer with satellite-link will routinely help in expediting the matter. Now of course mobile phone to remain constantly in touch with different officers, coroners office and lawyer will also make the collection of evidence absolutely reliable

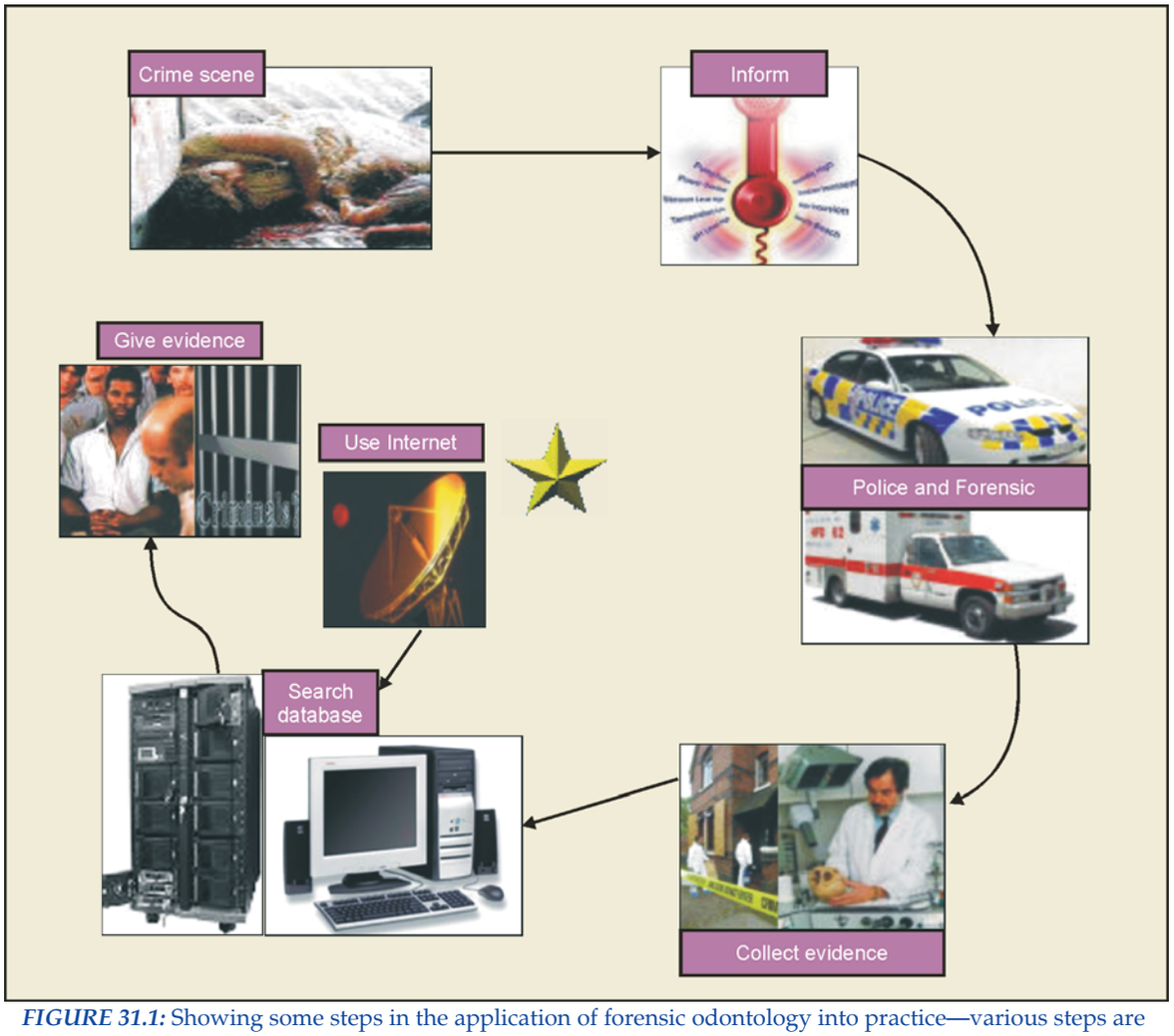

FIGURE 31.1: Showing some steps in the application of forensic odontology into practice—various steps are shown from various sources (2004)

and any doubts or questions can be verified against the police data bank or legal matters clarified with lawyers. Rarely of course the dental officer may have to contact the next of kin to announce the bad news of the fatality, accident or occurrence; from the site itself.

d. **Data recording** must be done on pre-formatted sheets so that no point however small is missed. Using a mini-recorder or an assistant will help in the keeping the professionals hands free. All measurements usually to be made in International units and in photography such that a metal or plastic scale is clearly seen in background.

e. **Shifting the deceased to the forensics area**: Once the body is shifted to the forensic medicine department it's a good idea to team up with the general pathologist/ coroner/ police pathologist and observe the whole surface meticulously. Look for bite marks, nail scratches, and if the saliva is suspected to be present over some body surface try to collect it in proper storage bottles. Other fluids like semen may rarely be present in the oral cavity, its collection must be done as quickly as possible.

f. **Embalming the remains** of the medical and dental autopsies and seeing that next of kin get them so that proper burial could be done as per the personal beliefs of that person or his dependants.

g. **Multiple radiographs** Intraoral-periapical type of the whole mouth may be taken, if the OPG machine is available, then the full jaw radiography must be attempted. It is a good idea to mount all the X-ray films and photograph making them multiple copies since many police and court departments may need multiple copies and also the original radiograph may deteriorate with time if improperly fixed (Due to overloading of the legal system even the criminal cases take years in India to get resolved).

h. **Making the impressions** of the bite marks, making the upper lower casts of the cadaver and intraoral photography; preferably using a digital fiber optic camera would be ideal.

i. **Comparison of the collected data** with known data bases like police database, passport information base or armed forces databases will help in comparing and establishing identity. Contacting the local dentists will be first line of search against any peculiar type of denture or crown and bridge preparation, the logs of the local dental labs would be able to furnish some clue as to which dentists are doing a particular type of work.

j. All the data collected should be filed physically and electronically into a computer system so that it is a back up. Most Indian courts and Judges may not accept electronic or digital data and hence paper files and photographs are a must at least for now. But scanning of all the photographs, X-rays and other forms will help to disseminate the information if required by International agencies like Interpol or police departments of the other countries. (Global criminals are becoming common!) (Fig. 31.2).

k. **Presentation of the evidence in court** on invitation of the attorney generals office/police or concerned legal department should be made. In such a presentation no jargon must be used since the opposition lawyer and judge should be able to comprehend your language.

CLASSIFICATION

Forensic dentistry study is classified as:

a. Civil
b. Criminal
c. Research

Civil

- Mass disasters like airline accidents, earthquakes or train accidents require identification of the victims in advanced stage of physical destruction
- Malpractice and different types of fraud
- Neglect where damage may be sought
- Age assessment of the individual like in cases of teen marriages in absence of any birth documents etc
- In case of accident victims who are suffering from Amnesia who may have to be identified
- Where long lost heirs emerge after many years, to claim substantial inheritances and are challenged.

Criminal

- Identification of the persons from their dental remains alone in cases of homicide, rape or suicide
- Detailed analysis of the bite marks
- Rugoscopy
- Lip print analysis.

Research

- Training seminars between the medical forensic, dental departments and the police so that immediate application of oral medicine knowledge in the justice section can be encouraged.
- Advanced courses like MDS in the field of forensic odontology seems to be urgently required.
- Dentists working for government or police departments must be immediately deputed to the Universities abroad like University of Adelaide in Australia where a course in forensic odontology is available.

MAINTENANCE OF DENTAL RECORDS

"Ignorantia juris non exusatus" is a famous legal maxim which means ignorance of law is no excuse to condone a crime. Every citizen should know the law of the land. Dentists are no exception. They should possess a working knowledge of the laws relating to practice of the profession for his patients and his own protection. Following are the absolute essentials in practice setting-

Maintaining the proper patient dental and medical health records every dentist must keep a written or electronic record of the patients details.[3] If computer record

is maintained then in addition to the details on the hard disk, one print out with patients' signature should form a part of the filing. Use of a visual system in which dental chart is portrayed with diagrams of deciduous and permanent teeth and color markers are used to denote caries fillings and missing teeth.

The radiographs, the IOPA and the OPG and others should be processed correctly with adequate washing, otherwise they turn yellow and then brown with age and become useless as records as they cannot be read. Proper cover for the radiographs with salient history details; and of-course now with the digital radiography (RVG-radiovisiography), storage of millions of radiographs on hard disk is a reality.

- Storage of the upper and lower plaster casts should be the prudent practice. Proper storage rack duly labeled on date basis or in alphabetical order can help in timely retrieval if needed.
- All the patients' medical health records, lab reports and medical consent letters from the physician should be filed and available in original with dentists record. A xerox copy may be given to the patient if requested.
- In a review of cases from 1983 to 1992 in Sweden, Borrman H et al[7] 1995 emphasized the importance of ante-mortem information for purposes of personal identification. Information on dental characteristics, normal anatomical findings and restorative treatment was complete in 68 percent of the cases. Registration of previous therapy was missing in about 94 percent of the records. In India the keeping of complete records is yet to become a routine practice, most dentists maintain only a appointment dairy. This practice needs to be changed and the new graduates should take a lead in maintaining a proper and detailed dentomedical record of each patient.
- Nobody except the patient and the dentist has the right to see the confidential medical records, and once he is privy to personal information like patient visiting prostitutes (sex workers) or STD record of past, homosexuality, etc. even police cannot take the records of any dentist except if ordered by a Judge who sees reasonable cause to check the records (for furtherance of justice).

CONSENT

Definition—Research—Medicolegal—Minor's consent—Major's consent.

According to the section 13 of the Indian Contract Act, " consent" means " Two or more persons are said to consent when they agree upon the same thing in the same sense. " Free consent is defined under section 14 of the said Act as under. Consent is said to be free when it is not caused by

- Coercion (section 15) - Undue influence (section 16) - Fraud (section 17)
- Misrepresentation (section 18) - Mistake (section 20,21, and 22) and is given of ones own free will Dalal JM[4] .

The dentist should always explain to the patient the details of the procedure to be carried out in a language they understand and in presence of a witness. Local language consent forms should be kept ready and thumb impression may be obtained in case of persons totally unable to write. The witness should affix his or her signature immediately next to it identifying the persons thumb print and also mention the fact that details of the treatment and its side effects if any have been mentioned.

Please note that forms given are merely indicative. Consulting ones personal lawyer prior to designing your consent forms is a very good idea! Use of local language and its English translation is recommended for everybody's convenience.

Consent for Research Work

1. The local ethical committee of the university hospital/ institution must approve any research on the human subjects.
2. Individual consent must be obtained from each subject in presence of one witness from the patient's side and one from the institution. The investigator or his representative must explain clearly the objectives of the research project; the fact that the procedure tested is not standard treatment and the details of the side effects in simple understandable terms.
3. Consent:
   I……..aged……..residing at ……
   agree to take part in the research project ".————
   ————————————————————" I understand that the treatment/procedure used is not one used

routinely and that I can discontinue the said procedure at any given time. This discontinuance will not affect my any other treatment in this institution or any other institution. I give this consent of my free will and wish. I have been explained this matter in the language I understand in front of one of the witnesses.

Signature of the patient	Investigator
Witness with the patient	One other hospital employee/witness
Dated:	Place:

Consent for Medicolegal Purposes

I (name of the suspect) aged about ……..……. residing at……do here by grant Dr.…….. or qualified technicians under his direction to examine, photograph, take impressions of my oral region and teeth.

I give permission for the taking of saliva samples and blood samples or any other sample deemed necessary for the investigation (semen, sweat or hair) (cross out that which is not being done).

I understand that this is in investigation of the accident/death/injury of ……..…… (Name of the victim)

I am co-operating of my free will and in my complete consciousness, without and undue influence.

This is in a language that I can understand and explained to me in front of my friend, family member, volunteer, interpreter (cross out which is not applicable) whose signature is affixed below as witness no 1.

Signed (suspect):	Doctor's signature
Signed witness no 1:	Witness no 2:
Dated:	Place:

Before going for a medicolegal examination a court order or legal consent from the suspect is necessary for these reasons

1. Suspect may file personal assault charges against the forensic dentist.
2. The evidence of this nature which, is obtained without consent, may not be admitted in the court of the law.

Consent in Case of Minor or Lunatic (Mentally unsound) Patient

Minor is defined as a girl who is under 18 years of age and boy who is below 21 years of age.

Lunatic patient is one who is certified by a psychiatrist, as having a particular psychotic diagnosis that the law admits makes the patient not responsible for his behavior. Such patients need hospitalization or need strong medication with constant supervision as safeguard for their own health.

I……aged about ……. and the parent/legal guardian of the minor …….. who is aged about ……do declare in my full consciousness that my child/my ward may undergo professional dental examination/ investigations and any procedures which are normally done in such instances.

Do declare that Dr……has declared him/her as psychiatrically incompetent to be considered responsible for his own actions (Copy of the medical certificate is attached).

Please delete the sentences not applicable

I further state that the dentist and anesthetist have explained to me the fact that side effects and consequences of the said treatment rendered, and these will not make them liable for any prosecution.

Parent—Legal guardian	Dentist
Witness # 1	Witness # 2
Dated:	Place

Consent for the Major Patient

I……..aged ……...residing at ……have decided of my own accord to undergo this dental examination/investigation/and treatment and related procedures at .....…… ……..(Name of the clinic/ hospital) by Dr……. or his appointed assistants. I have been duly explained the side effects of the same and will not hold them responsible for any consequences as regard with the procedures.

I agree to co-operate fully and realize that failure to do so will result in less than optimal results.

Dated:	Patients name:
Place:	Dentist's name:
Witness # 1	Witness # 2

TORTOUS LIABILITY

The term Tort means wrong or defined as civil wrong. It is an established principle under tort law that harms suffered

voluntarily do not constitute a legal injury and is not actionable. This principle is embodied in the maxim "Volenti non fit injuria"

A person cannot complain to the chances of which he has exposed himself with knowledge and free will this principle is founded on good sense and justice. The maxim applies to the consent, which runs the risk of harm that would otherwise be actionable.

BITE MARKS

The first man on earth 'Adam' committed the sin of biting an apple and was punished by God. Despite this fact, the man on earth never realized the importance of bite marks and its proper utilization.

Just as no two finger prints are alike, neither are two bite marks. It has been aptly described "While the criminal may lie through his teeth his bitemarks reveals all and do not lie"

The American Board of Forensic Odontology ABFO [5] was organized in 1976 under the auspices of the National Institute of Justice. ABFO Bitemark definition is as follows: A physical alteration in a medium caused by the contact of teeth. A representative pattern left in an object or tissue by the dental structures of an animal or human.

Bowers[1] 1996 has emphasized the need that for unequivocal identification of the assailant the matching of DNA testing from saliva and the bite marks matching both should be positive. He jokes that if both don't match if will be difficult to convince the judge that there were two assailants, one bit the victim and second who spat!

Teeth are the most non-destructible parts of the body; they are likely to remain following the loss of soft tissue by putrefaction or dissolution. Their individuality of shape may be the only way to determine identity when other features have long disappeared. The teeth that are present may have unusual characteristics of size, shape or surface contour and they may be affected by environmental conditions reflecting the lifestyle of their owner. The arrangement of the teeth within the upper and lower arches may be regular or irregular so that people who have never attended a dentist may still have sufficient idiosyncrasies in their teeth to enable them to be identified.

In studying the bite marks it is important to start with the wound. If the wound is fresh then the saliva samples should to be taken from the site. The photography of the marks and making of proper impressions using rubber base impression materials is immediately recommended. Variability of bites can be particularly striking in cases where multiple bites are present. If the victim is alive and able to give history then classification of definitive marks, Amorous marks or aggressive marks can be made.

Sansare K (1995)[6] states that the advances in dental material,laboratory techniques and improvements in scientific and photographic technology, have made the evidence from different dental tissues more dependable and reproducible.

Sweet D and Bowers CM (1998)[9] have compared the computer-based, radiographic, xerographic and hand-traced methods. The computer-based production method was determined to be the most accurate of those studied. It produced accurate representations of the biting edges of the teeth in an objective manner. The radiographic method was determined to be more accurate than the xerographic method with respect to tooth area measurement. The hand-traced methods were determined to be inaccurate and subjective. It is recommended that forensic odontologists discontinue the use of hand-traced overlays in bite mark comparison cases.

Aboshi H et al (1994)[10] found that a suspected arsonist had left his characteristics bite marks on four pieces of cakes in Mount Gambier in South Australia. These marks were compared with those of his actual dentition using a computerized imaging analysis technique. This established that it was the same person with a large degree of certainty.

The uniqueness of the bite marks can be determined on the following pointers:

1. Arrangement of teeth
2. Teeth missing from the arch
3. Supernumerary or specially shaped teeth, like mesiodens, microdontia etc.
4. Hereditary conditions like flourosis, distinctive malocclusion, hypoplastic teeth etc.
5. Radiographically unique positions of impacted third molar or any other tooth like an supernumerary would

probably give a clue to the identification if ante-mortem radiographs are available for comparison.

6. Patterns of abrasion, attrition and erosion must be looked for.
7. Uneven or accidental wear like needle holding, nail holding in between the incisal edges in professions like tailors or carpenters or an asymmetric pipe mark on the mandibular teeth on suspect known to be pipe user.

Class Patterns of Tooth Types

The incisal or occlusal portions of the teeth generally cause the injury pattern in bitemarks in tissue. The injury pattern seen in bite marks generally reflects the basic shape of the incisal or occlusal area of the tooth type in question.

Incisors leave a rectangular shaped mark. Beveled markings are more common, sometimes with perforations at the incisal angle areas. Canines cause the triangular markings with apex of the triangle towards the labial and base towards the lingual. Premolars cause single or dual triangles with the bases of the triangles facing each other or coming together as a diamond shape.

Molars rarely leave bite marks and when they do they leave unique quadrilateral markings.

Individual Characteristics

Deviations from the standard class patterns reflect themselves as individual characteristics. The recognition of class characteristics and consideration of these in bite mark analysis are vital in drawing conclusions. A labially placed canine or buccally inclined first premolar is the examples of individual characteristics in addition to the seven points mentioned above.

Methods of Bite Marks Comparisons

Computerized Method

Use of digital cameras to record the dental arch arrangement of the corpse and its subsequent comparison with ante-mortem records using specially designed hardware is currently the latest method of bite mark comparison.

Nambiar P et al (1995)[12] have recommended the use of an interactive shape analysis computer program ("SCIP"). They also introduced the concept termed as similarity index (SI). They opine that if reasonable number of reference points have been registered then matching of an offender with bite mark is actually possible.

Photographic Method

Intraoral photographs of the upper and lower dentition are compared with the photographs of the bite marks. If possible the actual position of the body at the time of bite is reconstructed so that alteration in the skin fold and its impact on the photographic picture may be taken into account. Polaroid camera should be a standard kit for the forensic dentist so that immediate prints in absence of processing facilities may also be possible.

Impression Method

Full mouth impressions of the suspect are made and then compared with the rubber based impression obtained of the bite marks on the food, skin or other substances.

Making of acrylic teeth casts and then using it to compare with the bit-off part of the victims ears is a beautiful case reported by McKenna CJ et al (1999)[13].

Manual Overlay Method

The suspect bites on a carbon paper (typewriting duplicating paper) with a clean white sheet below it. A small cardboard can be used to protect the upper and lower bites. Then the paper is opened up and the relative positions of the incisal and occlusal marks of teeth may be studied. Even though Sweet and Bowers [9] have cautioned that manual method of bite mark evaluation is subjective, sometimes in a rural setting in Indian the computerized and other sophisticated techniques may not be available.

Palatal Rugae Pattern Evaluation

Many studies have shown that palatal rugae are as unique as the finger prints.

Limson KS and Julian R[19] have demonstrated in their computerized study the efficacy of the rugae analysis. They analyzed 250 subjects and created antemortem records and based on this they used an indigenously designed software and found that the sensitivity for correct match was 0.93 and specificity of 1.00 and had success rate of 92 to 97 percent in the matches with digitized rugae pattern.

SALIVA DETECTION

a. **The Laser system of detection:** The saliva left on the skin, food or in a spittle may be very crucial in identifying the perpetrator or criminal. Springer E et al (1994)[15] have mentioned that Nd:YAG laser emitting at 266 nm can be used to detect dry stains of blood, semen and saliva in scene of crime so that material can then be collected assiduously by the investigator.
b. **ELISA method for blood grouping:** Komuro T et al [16] 1995 have standardized a method using enzyme-linked immunosorbent assay (ELISA) for identification of the blood groups ABO from the collected samples. A horseradish peroxidase conjugate in combination with the use of monoclonal antibodies using a fully automated system performed this ELISA.
c. **Comparing evidence obtained from saliva and confirming it with hair follicle evidence**: Allen M et al (1998)[18] mentioned that human hair and saliva are frequently left behind by the perpetrators and is the commonest evidence material. The authors have reported a 90 percent certainty rate by using a amplification system for detection of sequenced mitochondrial DNA(mt DNA). In three robberies this method was demonstrated successfully.

NON-ACCIDENTAL INJURY

The changing sociocultural milieu in India where divorce rates are soaring and more and more women have to go to work and to fend for themselves, the traditional fabric of family is getting torn apart. It's the innocent children who pay the price. They get beaten up, shaken or worse sexually abused by adults who are their caretakers and venting their frustrations.

Dentist is one the health care workers who may first see the child who is a victim of such crimes. He must be familiar with what is happening in the society around him and be ready to report to the police and foster care facilities run by Department of Child Welfare, at a local level.

The term "battered-child syndrome" was coined by Kempe in 1962 but now-a-days terms like non-accidental injuries and shaken baby syndrome (SBS) are found in literature. Shimura T et al (1994)[2] analyzed 8 cases of battered children and found this clinicopathological entity in the central nervous system is characterized by retinal hemorrhages, subdural and subarachnoid hemorrhage. They have emphasized that use of CT is extremely sensitive for detection of these changes.

Alexander RC (1995)[17] states that neglect, physical abuse and sexual abuse are three aspects, which may be considered collectively as child abuse. Continuously berating an individual child by parent or caretaker can also constitute a relatively newer entity termed as emotional abuse that can wreck the child psychologically. In his series almost 50 percent of the case were of child neglect, 21 percent involved in physical abuse and 11 percent had overt signs of sexual abuse. The emotional abuse was detected in less than 3 percent children.

In developed countries with an efficient social security system the under-reporting of the child abuse cases continues. In India the problem is still worse. Even in households where it exists the refusal to accept it as a problem can lead to serious consequences for the child. Serious efforts need to be made by all health care personnel in conducting more research and educating the women. This will help in evolving locally relevant prevention tactics. We need to wean away the parents and many teachers from *spare the rod and spoil the child philosophy* and bring in more creative teaching methodology.

Dentist must look for following 'hot' areas to suspect the child abuse case:

1. Very shy withdrawn child with multiple bruising and broken teeth.
2. No proper history is given or dentist feels that parent or caregiver is lying about the incident.
3. A pattern of multiple and healing fractures is radiologically noted.
4. Child is coming from a family where parents are in social turmoil of separation, divorce or there is ongoing alcohol or drug abuse in the family.
5. Basically dental surgeon must use healthy common sense, focus on the social and family history of the child and try to seek help for child from government agencies, medical-social workers and other child-care agencies if the situation seems to be getting out of hand.

DENTURE MARKING

After the tragic earthquakes of Bhuj and Latur in India, the Army jawans and the aid workers have found many corpses with their dentures on. But since in India no uniform code for labeling or marking of dentures prevails this evidence was lost to the investigators.

Personal identification from dentures is one of methods used in forensic odontology.[11] The partial denture is easily matched to the missing teeth but a complete set of dentures with on other markings was matched using CT imaging technique. The alveolar ridge and the denture surfaces were matched using photography and CT image photographed for superimposition. They found this method extremely useful.

Borrman HI et al [7] 1999 have commented that in Sweden as per the recommendation of the National Board of Health and Welfare all dentures must be permanently marked with a stainless steel band incorporated into the acrylic containing patients birth date and a special identifying number. A survey from the Nordic countries has shown that if denture marking was in general use, the contribution to the establishment of identity by forensic odontology in cases of fire would increase by about 10 percent.

In India the Indian Dental Association must send a directive to all the laboratories that when making dentures some form of identification like full name and address may be incorporated on a thin aluminum sheet and then embedded in the denture as a routine.

USE OF PRINT MEDIA AND INTERNET IN FORENSIC ODONTOLOGY

Use of dental journals and print media are one the most important sources of the ante-mortem information locators. If a dead body which refuses to be identified by conventional means is found, usually the police take full mouth radiographs and intraoral photographs and publish them in media which the dentists may frequently read in a hope that one of the dentists will recognize the fillings or the dental configuration and help in identifying the corpse. According to Alt KW and Walz M[14] 1999 who evaluated 177 cases published between 1975 and 1995.They showed that only 3 percent of the cases were identified from recognition in the dental print media. This figure is dismally low. But with the wide spread use of internet, digital X-rays and digital intraoral radiography and rapid online data transfer, a day has come when the

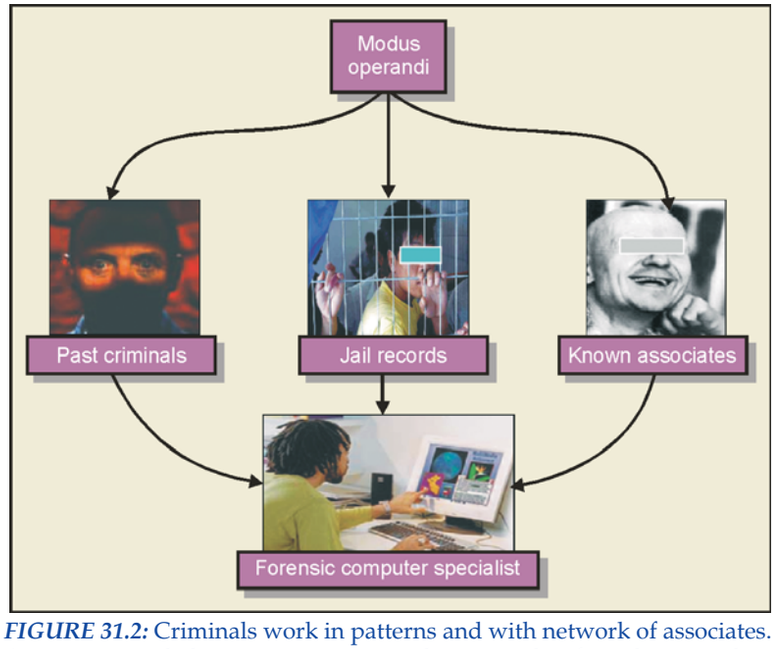

FIGURE 31.2: Criminals work in patterns and with network of associates. A computer can help us to see patterns, and compare data from disparate data bases and predict where the crime may occur. (Bailoor DN at forensic odontology workshop at Kshema Deralkatte, Mangalore on 19-20 (June 2004)

forensic odontologist will get international cooperation from the internet for the recognition of that unidentified body.

CONCLUSION

As the crime in Indian scene is on the increase, the justice system needs different tools to fight the criminals and protect the innocent. One such tool is forensic odontology. In India it is an underused and underdeveloped specialty and we all need to work to develop a body of personnel trained in this discipline. Another discipline all the dental surgeons should develop is the meticulous keeping of dental records. Without proper ante-mortem records to compare many of the forensic dentistry steps become redundant.

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32 Biopsychosocial Model of Illness and Oral Medicine

INTRODUCTION

Biopsychosocial model of illness was first proposed by Engel in 1977 and later modified by many workers. This model suggests that any illness is caused due to the interaction of three factors: the biological, the psychological and the social. The first example that comes to mind is of HIV infection. The biological agent is the virus and psychological stress and loneliness causes the person to indulge in drug habit or promiscuous sex, and the social stigma of AIDS may drive the patient to suicide. In this disease all three factors are easily identified.

- ***Psychology:*** It is that specialty of the science, which studies the working of the normal mind, the perceptions, sense organs, the personality formation, learning, anger, stress etc. Sperling [1] calls it the science of individual behavior and experience. Understanding the fundamentals of psychology also help in the superior emotional intelligence.[13]
- ***Psychiatry:*** It is that branch of medical sciences which diagnoses and treats the diseases of the mind, and also tries to elucidate the disorders which are in the twilight zone of mind and body i.e. the psychosomatic disorders. Sperling [1] defines it as the study and the treatment of mental and emotional disorders.

Figures 32.1 and 32.2 shows the comparison of the older medical model of illness with newer Biopsychosocial model of the illness. The importance of psychological and social factors in the causation of disease is very apparent in disorders like HIV infection and the stress related problems like tension headache etc. The impact of stress on immune system can lead to various changes and the whole gamut of diseases, which respond with difficulty to regular treatment.

Biopsychosocial Model of Health

Engel GL (1997)[21] mentions that too much reliance upon self-report inventories loses the essence of the doctor-patient relationship. This article emphasizes the importance of the medical interview not only as a human encounter but also as a rigorous instrument to better understand the patient and help explain the data that the patient presents. Subjective experiences such as sadness, grief, and fear are not soft signs but essential elements of a patient history. His seminal paper on the biopsychosocial model became an organizing principle for psychiatric education in medical settings. It is the challenge—yet the reward—of the physician to empathically make meaningful connections between the patient's life history and presenting problems to diagnose the difficulties with which the patient presents.

Berman ME (1997)[19] states that Biopsychosocial models are multidimensional explanations that attempt

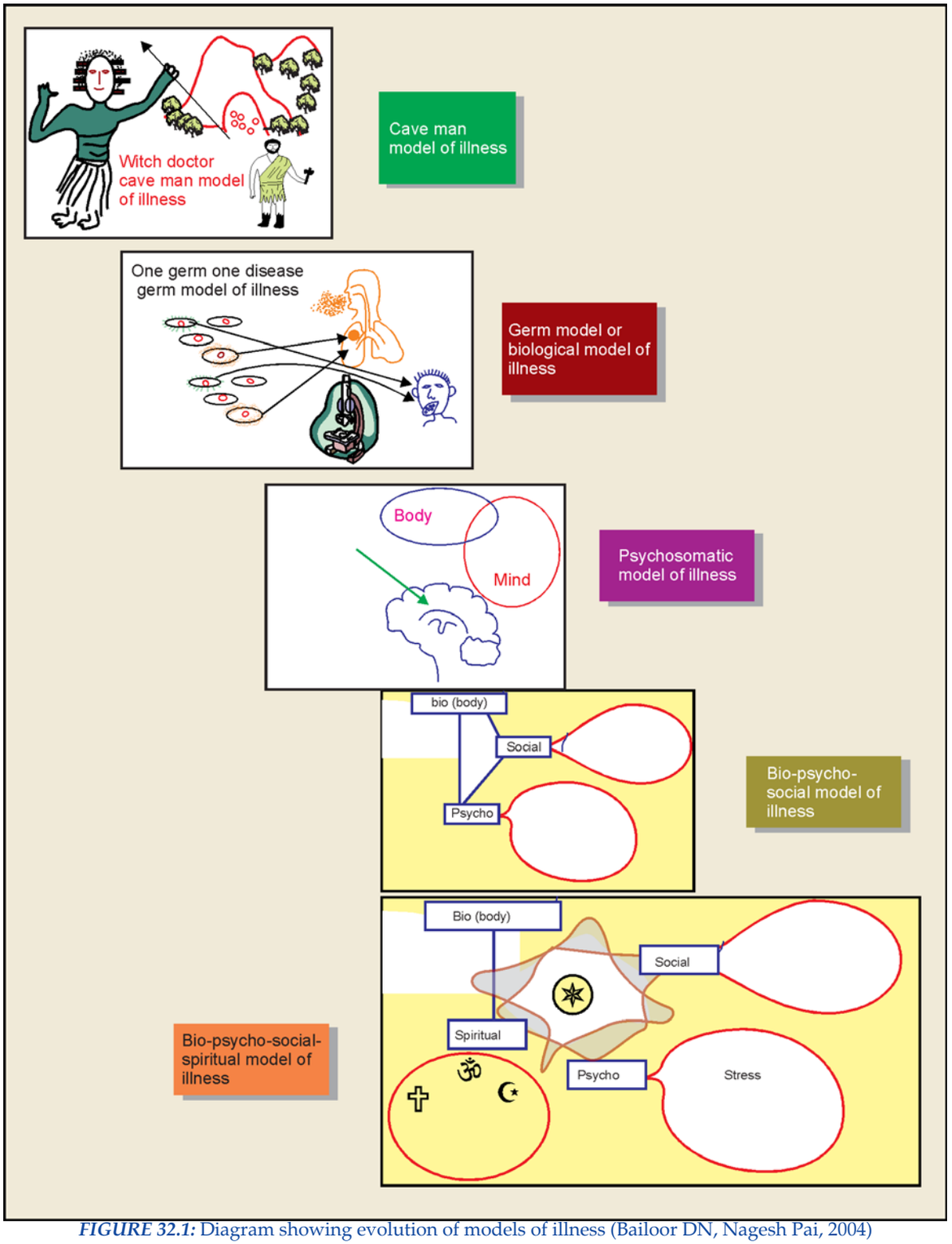

FIGURE 32.1: Diagram showing evolution of models of illness (Bailoor DN, Nagesh Pai, 2004)

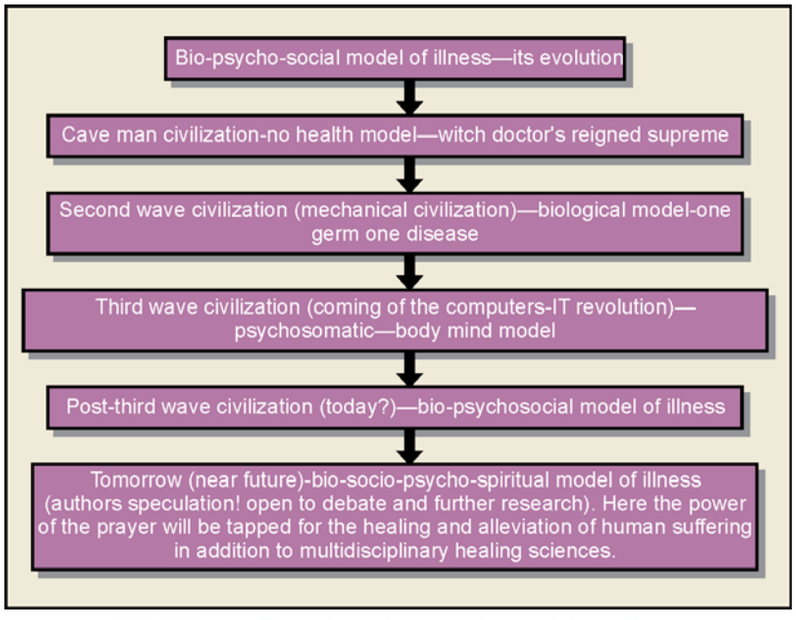

FIGURE 32.2: Flow chart showing the models of illness (Bailoor DN, Nagesh Pai, 2004)

to provide a framework for understanding how biologic contextual, and individual difference variables combine to influence human behavior.

Schmidt H (1998)[20] describes integrating the teaching of the basic sciences, clinical sciences, and biopsychosocial issues in medical education as part of the curricular reform efforts initiated by schools that participated in The Robert Wood Johnson Foundation's Project "Preparing: Physicians for the Future: Program in Medical Education." The author strongly recommends the integration of sociological and psychological sciences in the health care milieu and training of the future batch of oral physicians and dentists.

Bartz R (1999)[17] states that the biopsychosocial model has been a cornerstone for the training of family physicians; however, little is known about the use of this model in community practice. He opines that the cultural factors are also important when the doctor practices in a different socioculture milieu. We need further understanding of the inter-relationships between physicians' clinical environments, knowledge of patients, and theories of disease

Previous studies have established a powerful relationship between socioeconomic position and health. Fiscella K [18] 1999 mention that lower family income was significantly associated with poorer health status, greater psychological distress, more family dysfunction, less social support, more behavioral risk factors, higher rates of obesity and uncontrolled blood pressure.

The face and the mouth are special areas for a person, since face identifies the self. The circumoral musculature give sensual satisfaction from childhood (suckling the mother's teat); going through transient gratification of thumb sucking, lip sucking (oral habits); in adolescence results in pencil sucking, chewing and finally maturing in adulthood into an satisfying erotic zone for sexual stimulation, arousal and satisfaction.

It is then no small wonder that many of our psychological and psychiatric manifestations make an appearance in this region.

The primary health care provider the dentist must remember that 4 groups of patients may present with symptoms in the dental clinic:

I. Emotionally fit individuals who are under stress.
II. Those with transient emotional illness, such as an anxiety state or depression in addition to pain.
III. Patients with persistent hypochondriasis
IV. Those with frank psychotic disorder.

Following orofacial disorders have a direct biopsychosocial etiology

1. TMPD (Temporomandibular pain dysfunction syndrome)
2. BMS (Burning mouth syndrome)
3. AFP (Atypical facial pain)
4. Xerostomia—Dysgeusia
5. Wide gamut of orofacial diseases which have Biopsychosocial contribution or triggering factors in the individual or the family.
   - **i. RAU** (Recurrent Aphthous Ulcer): McCartan BE et al (1996)[22] measured anxiety and salivary cortisol in two groups of patients with recurrent aphthous ulceration. One group of patients had persistent aphthae (Group 1) and the others had been relieved of their aphthae following correction of detected hematinic deficiency states (Group 2). Anxiety was measured using the hospital anxiety and depression scale and radioimmunoassay of salivary cortisol. They concluded that stress may play a role in the aetiology of recurrent aphthous stomatitis, particularly in patients who have an underlying anxiety.
   - **ii. OLP** (Oral lichen planus)—This mucocutaenous white lacy lesion was said to be of unknown etiology. Immunologists determined that autoimmune reactions were primarily responsible. Today behavioral sciences have determined that stress and psychosocial changes can cause the body to interact in an autoimmune way. Thus apart from cortico steroids and immunomodulators, the antianxiety drugs and tranqulizers have also been used by dentists around the world.
   - **iii. ANUG** (Acute necrotizing ulcerative gingivo-stomatitis): This infection caused by fusospirochetal organism is seen in poor regions of the world like Ethiopia, Somalia, and Bangladesh. The children and young adults who suffer from this infection are under serious stress both psychological and nutritional. If untreated this disorder frequently complicates into Noma, which is a gangrenous destruction of facial tissues. Second world war saw many soldiers suffering from this infection, due to war stress and was called the "trench mouth".
   - **iv. Anorexia nervosa (AN)**: It is a disorder of teenage and young girls. Modern civilization, fashion magazines and television showing models who are bony skinny have spurred the young women into seeking unrealistically thin figures by dieting too much and frequently by persistent self-induced vomiting. Thin pale women go around complaining that they are overweight and are obsessed with not having food. They are depressed and frequently withdrawn and given to mood swings. Dentists may see such women with multiple chemical erosions on the palatal aspects of maxillary teeth and hypersensitivity.
   - **v. Hysterical dysphagia**: Dysphagia means distressed swallow or disturbed swallow. It may have organic cause like tonsillar enlargement, pharyngeal tumor or carcinoma of esophagus. Sometimes no pathology is detected and patient has difficulty in swallowing liquids but no difficulty in swallowing solids. This seems rationally impossible and more often then not the cause is psychogenic.
   - **vi. Bruxism**: Whenever frustrations of modern life catch up with us and we are unable to do anything, the teeth are held together and ground against each other. The muscles like masseter become tense and painful. The teeth show flattening of cusps due to wearing away. Some persons have such serious bruxism that people sleeping near them get awakened. There is severe jaw soreness when they get up in the morning.
   - **vii. CFS** (Chronic fatigue syndrome): This is another disorder highlighted in so-called civilized society where apparently without any disease, patient loses interest in job family and surroundings and appears to be tired all the time. Post-viral fatigue syndrome is a term used by some investigators from the American Universities but most European references emphasize on the psychiatric etiology. High levels of stress, frequent change and mechanical life have all been implicated in this syndrome that is under research and scrutiny.
   - **viii. MS (Munchausen Syndrome, MS by proxy)**: This syndrome is characterized by patient demanding complicated investigations for imaginary diseases. This may be considered one of the complex forms of

hypochondriasis. Mother of a young patient who is persistently brought to doctor for insignificant illnesses with demands of very expensive and far out investigations may exhibit sometimes MS by proxy.

ix. **Odontophobia and other phobias**: Abnormal fear of dentists and dental procedures is termed as odontophobia. This aggravated fear prevents patients from seeking dental care in spite of having financial means and urgent need. Thus patients who are seen with advanced dental neglect should be viewed with suspicion for odontophobia.

x. **BCS (Battered child syndrome)**: The term non accidental injuries (NAI) has also been used to denote children who appear in trauma out patient or dental clinic with severe physical damage and mutilation. This is mostly in hands of those very people who are supposed to take care of them. Father, mother, foster parents or relatives usually use excessive physical force and beatings to vent their frustrations. Histories of alcoholism, drug abuse, prostitution and low socioeconomic status have been found in most cases. Dentists can note fractured mandible, broken teeth and bruises in unnatural places with improper explanations, about how it happened. Full body radiography can frequently reveal multiple fractures in stages of healing. Such children need to be referred to social cells, related government agencies and counselors for family treatment.

xi. **Factitious ulceration** (Stomatitis artefacta): Self-inflicted wounds in the oral mucosa by neurotic individuals. Some of them use pins, nails or caustic agents to create ulcers. These lesions are considered as the oral counterparts of dermatitis artefacta.

xii. **Morphodysphoria**: It is an obsession that "I don't look good". I need surgery to look better.Patients who are average looking or even fairly good looking keep coming in for cosmetic surgery or orthodontic advice. They have already self-diagnosed themselves as ugly and needing emergency cosmetic care. Many times it may be the only finding in absence of any psychotic features.

Scheutzel P (1996)[15] has mentioned that dental erosion which is unexplained in dental patients can alert the clinician to the fact of GERD gastroesophageal reflux disease or other causes of persisting vomiting like anorexia nervosa. The etiological factors for the Biopsychosocial disturbances should be kept in mind when dentist performs the other treatments.

Jackson JL et al (1998)[14] have mentioned that in the patient history record if 4 S is seen i.e., S = stress, S= somatic symptoms, S= status of health (continuously poor) and S= symptoms severity varying unpredictably. Then screening for mental disorders should be more rigorously done. In our department we normally administer HAD (Hospital anxiety and depression inventory) to quantify the level of depression and anxiety.

TEMPOROMANDIBULAR PAIN-DYSFUNCTION (TMPD) SYNDROME

TMPD syndrome is a biopsychosocial disease which is diagnosed by criteria given by Laskin [2,] namely pain in the preauricular region, clicking and popping sound, tenderness in the muscles of mastication specially in the morning, and the absence of any organic joint disease in various imaging modalities used.

According to the study done on South Indian populations by Bailoor, Kumarswamy et al[3] the ratio between male and female was 1:6 and the dominant age was 20 to 30 years. These figures seem to agree with the studies on Caucasian populations by Helkimo[4] (Studies of Butler et al, Carlsson et al in Helkimo).

Gold and coworkers [5] compared the incidence of four different psychosomatic disorders in 135 TMPD patients with equal number of normal populations. Their findings are shown in Table 32.1.

Table 32.1: Showing the increased incidence of four *Psychosomatic disorders in the patients with TMPD*

Psychosomatic disorder	*TMPD group*	*Control group*
Low back pain	64%	23%
Aphthous ulceration	18%	2%
Irritable bowel syndrome	48%	27%
Asthma	12%	5%

The management of the TMPD is normally discussed as a step wedge, see the TMJ disorders chapter.

It is a good idea to administer hospital anxiety and depression (HAD) inventory and determine the level of

anxiety and depression in the patients. Use of General health questionnaire (GHQ), hamilton anxiety questionnaire and in some cases Personality assessment is also indicated using the 16-PF, or MMPI (Minnesota multiphasic personality inventory)

Here we divide the treatment modalities into.

Pharmacotherapy: Aspirin, antidepressants, and muscle relaxants and anxiety relievers all could be used to initiate pain relief.

Dental Rx modalities: Occlussal splints, occlussal adjustments.

Physiotherapy: Hot compresses + Pharmacotherapy
Cold application + Jaw exercises

Psychiatric modalities: Counseling, minor tranquilizers, antidepressants, etc.

Other modalities like: Hypnosis, TENS, biofeedback, medication etc. all seem to give relief in selected cases. A good clinician rationally chooses his armmentarium amongst these therapies, Bailoor et al [3] undoubtedly proves that a joint therapy between the oral medicine specialists and the psychiatrist will make the management of TMPD complete and successful.

Gil IA et al (1998)[23] has indicated that chronic pain is the major complaint of myofascial pain dysfunction syndrome. It's a complex problem, which involves physical, psychological and social aspects. The etiology of TMPD is multifactorial and the multidisciplinary approach is essential for comprehensive treatment planning

Major PW and Nebbe B (1997)[24] have commented on Interocclusal orthopedic appliances of varied design and application that have been employed in the treatment of TMPD syndrome. These appliances provide the practitioner with a non-invasive, reversible form of intervention to manage the patient's symptoms.

Korszun A et al (1998)[26] have established that many of the stress-related conditions have comorbidity with temporomandibular joint disease. Chronic fatigue syndrome and fibromyalgia are two of the conditions, which showed co-existing disease. They blame the pathophysiologic basis involving dysregulation of the hypothalamic-pituitary-adrenal stress hormone axis in predisposed individuals. Of the 92 patients, of whom 42 percent reported temporomandibular disorders, 46 percent had histories of irritable bowel syndrome, 42 percent of premenstrual syndrome. Pawl RP (1999)[27] mentions that chronic neck pain can herald the beginning of myofascial pain disorders. Trauma and psychological factors have been implicated in chronic neck pain syndromes.

Molina OF et al (1999)[25] did extensive review of the literature, the analysis of two hundred and seventy-six CMD patients and compared their data to other studies allowed them to conclude that severe bruxers are more impaired by muscular and joint disorders as compared to mild and moderate bruxers.

BURNING MOUTH SYNDROME

Burning mouth syndrome is defined as a set of diseases, which may cause primary complaint of burning sensation without the presence of any local lesion in the oral cavity. Bailoor DN et al[6] have reported clinical characteristics of burning mouth in South India. They reported this syndrome in predominantly postmenopausal women. All had a degree of depression and were much better after psychiatric counseling and antidepressant therapy. In this group anemia was another important consistent finding. These findings seem to be in agreement with the findings of Browning, Hislop et al[7] and Grushka[8] who also confirm a strong association of this symptom with psychological disorders.

Schonberg[9] mentions that burning sensation goes on becoming worse as the day progresses, in menopausal females. This finding was not substantiated in the Indian study. Spielman A et al[10] in Haifa, Israel correlated xerostomia secondary to the anticonvulsant drugs, psychotropic drugs, and sedatives leading to burning sensation symptoms.

Other causes mentioned, B complex deficiency;[11] zinc level dipping in serum,[12] diabetes mellitus; and irradiation to the oral areas.

Burning mouth diagnosis can usually be established in elderly female patients by administering anxiety and depression rating scales with definite findings. Such patients are jointly treated with psychiatrists. Nutritional aspects of iron, folic acid, and B-12 together with zinc have been giving good results in our series (Raricap®, Fefol®

and Biostar®-one of these mentioned - one spansule a day for four to six months, antifungals, saliva substitutes, even creams of local anesthesia like Mucopain®, Hexigel® could be used in selected case).

Huang W et al (1996)[32] mention about a three step therapy, the first combination of antifungal, nutritional, and estrogen replacement. The second being long-term therapy with antidepressants, benzodiazepines, and clonazepam. And thirdly, topical capsaicin and laser therapy may be beneficial in some patients.

Grushka M et al (1998)[30] in their study of 30 patients with BMS found that Clonazepam may be helpful in burning mouth syndrome, in as much as 70 percent of patients. The starting dose was 0.25 mg per day and gradually increased by 0.25 mg until the symptoms abated.

Muzyka BC and De Rossi SS (1999)[28] state that burning mouth syndrome is a poorly understood oral condition. Women are affected by the condition seven times more frequently than males. Of all the etiologies psychological stress and menopausal syndrome appear to be closely linked to the etiological factors.

Virgili A et al (1999)[29] mention that causal factors for BMS may be psychogenic, systemic or local. They have implied that doing a patch test should be amongst the mandatory tests, so that in those patients in whom allergy is a factor, may be treated.

Scientific worker Fraikin N et al (1999)[31] suggest that postmenopausal women suffering from BMS are advised to take a hormonal replacement therapy even though no direct relation between BMS and estrogen blood levels has been proved. Its high prevalence among postmenopausal women has been proved by many studies throughout the world.

Glossodynia and glossopyrosis are terms often used inter changeably by clinicians. The former means painful tongue and latter denotes "tongue on fire". Both these labels also form a part of burning mouth syndrome.

Hautmann G et al (1996)[46] consider these terms as symptoms and International Association for Study of Pain (IASP) also seems to concur about this. Marxkors and Muller-Fahlbusch[47] have written a wonderful monograph on psychogenic denture intolerance. They found masked depression in 57 percent of the cases, neurotic etiology in 21 percent and schizophrenia in 3 percent. Feinmann CH et al (1984)[48] studied 160 glossodynia patients and submitted them to various diagnostic questionnaires. Ninety percent of this group was females and average age was 60. All these patients showed higher scores in both state anxiety and trait anxiety as compared to healthy controls.

The tricyclic antidepressants have an anticholinergic effect and may induce xerostomia aggravating the burning. Flouxetine, paroxetine and pimozide have been used either alone or in combination, by many clinicians with great success.

It is apparent that burning mouth patients present a diverse and complex group and the determination of etiology is a difficult and challenging problem.

XEROSTOMIA

Dry mouth or xerostomia is a symptom, which may be caused by varied etiologies such as—

- *Physiologic*—Anxiety, fear and situational problems.
- *Drugs*—Antihistaminic, atropine and analogues, tricyclic antidepressants, antiemetics, major tranquilizers, etc.
- *Pathologic*—Sjögren's syndrome, sarcoidosis etc.
- *Irradiation*—Rx of oral cancer etc.
- *Metabolic*—Diabetes mellitus, hypertension treatment etc.

  Dehydration—Diarrhea, persistent vomiting, anorexia nervosa
- *Psychiatric*—Depression, hypochondriasis.

Xerostomia and stress lead to another very puzzling disorder called as dysgeusia (which is defined as altered taste sensation).

Following are some causes for Dysgeusia:

- Depression
- Stress syndromes
- Glossitis—caused by iron deficiency—Vitamin B deficiency
- Zinc deficiency
- Tumors of the middle ear, temporal bone, parotid gland produces a unilateral anterior third dysgeusia.
- Tumor invading the lateral wall of the pharynx may damage the glossopharyngeal nerve leading to dysgeusia posterior third of the tongue.

- Intracranial lesions affecting nerves VII, IX, X, cause dysgeusia together with associated motor and sensory abnormalities.

Treatment of both these symptoms xerostomia and dysgeusia locally can be the use of artificial saliva, use of sialogogues, meticulous care of oral hygiene and use of flouride mouthrinses to prevent the rapid spread of caries.

Pujol T et al (1998)[33] found the prevalence of 9.7 percent in the population for xerostomia. There was a linear relationship between growing age and greater xerostomia. There was a strong association between xerostomia and the consumption of tranquilizers, antidepressant, anti-allergy, anti-hypertension and hypnotic drugs.

Zimmerman RP et al (1997)[34] have advocated 5 mg pilocarpine hydrochloride four times per day (q.i.d.) beginning on the first day of radiotherapy and continuing for 3 months after completion of radiation.They found no significant difference in patients who were continued after 3 months on this regimen.

Garg AK and Malo M (1997)[35] have stated that application of a fluoride gel daily can take care of dental caries and xerostomia needs to be combated by artificial saliva and sialogogues.

Field EA et al (1997)[37] studied 100 consecutive patients referred for investigation of oral dryness. They categorized the etiology as follows: A definite diagnosis of primary and secondary SS was made in 24 and 15 patients respectively. Other causes of xerostomia were: undiagnosed diabetes (3); drug-induced (11); therapeutic radiation (3); alcohol-related (3); psychogenic (15) and idiopathic (21).

Hamada T et al (1999)[36] mentioned that use of sialogogue, anethole trithione (AT) in 49 patients resulted in good salivation in 41 patients as compared to a control group of 45 patients of hyposalivation which were treated purely with artificial saliva. Only 9 in this group felt satisfied with oral lubrication.

ATYPICAL FACIAL PAIN

This normally refers to that pain which does not confirm to recognized anatomic pathways. Following clinical pointers are necessary before a practitioner can brand the pain as atypical.

- Meticulous clinical, radiographic and hematological examination should not reveal any abnormality.
- Rating scales should confirm presence of anxiety or depression.
- The nature and site of the pain will vary from time to time in subsequent visits
- The pain will cross over the midline and follow bizarre pathways.

Menopausal females, geriatric patients, and sometimes younger spinsters would combine this atypical pain with other forms of hypochondriasis like fits of fainting, weakness, nervousness etc. Once diagnosed this is an ideal disorder to be treated in hospital with help of Psychiatric social worker, psychiatrist and the dental surgeon. Counseling, short-term antidepressant medi–cation, and or minor tranquilizers normally provide good prognosis. At present even a CT scan is considered a basic test in order to rule any pathology lying in deep cranial areas.

Bailoor DN and Nillofer S[49] analyzed 21 cases of 14 females and 7 males and a strong relationship was noted between the atypical facial pain and depression and life stressors. The females invariably showed higher intensities of the varied symptomatology. Most responded to anti-depressants and multiple counseling sessions. Psychiatrist was invited to the Department of Oral Medicine and Radiology to evaluate certain severe cases who were reluctant to go the Psychiatry Department due to social phobia.

ODONTOPHOBIA

Odontophobia is severe aversion to dentist or dental treatment, which becomes so serious that patient's dental health goes from bad to worse. A milder form is called dental anxiety or dental fear, which can cause the patient to delay the treatment and cause the clinical situation to be aggravated. Sometimes patients have mild somatization problems, which can get focused on something like say the dental amalgam.

Shaw AJ and Niven N (1996)[41] have shown that hypnosis is a useful adjunct in management of dental anxiety in children and adolescents. They lament that it is unfortunate that it is not used more, since it would allow reduction in number of children getting anesthesia.

Moore R et al (1996)[42] studied effects of hypnotherapy (HT) and self-hypnosis training on extreme dental anxiety in adults aged 19 to 65 years. They reported great variation in ability of the patients to get hypnotized and found it a treatment option worth considering.

Locker D et al (1997)[38] found a close relationship between dental anxiety and blood/body injury. They used anxiety sensitivity index and Speilberger trait anxiety index. They also associated a group of dental phobics with agoraphobic symptoms and social interaction fears.

Gordon SM et al (1998)[39] found that 27.9 percent of the population reported fear/anxiety about dental visits, with approximately half of those reporting to be very nervous or "terrified". There was an inverse relationship between the frequency of dental visits and the proportion of respondents reporting themselves as very nervous or terrified.

Locker D et al (1999)[40] studied 1420 subjects and found that 50.9 percent, reported onset of dental anxiety in childhood, 22.0 percent in adolescence, and 27.1 percent in adulthood. Negative dental experiences were predictive of dental fear regardless of age of onset.

CANCEROPHOBIA

It is a persistent fear in the patients mind that they have contracted oral cancer. These are usually well-read, educated patients who constantly change their toothpastes, often use a multitude of mouth washes and are in general very finicky about the oral hygiene, but at the same time they go from dentist to dentist seeking reassurance that all is fine. This has been noted to be associated with depression.

Creagan ET (1999)[43] have given a wonderful review of the relationship between psychosocial, emotional and attitudinal factors and aggravation of malignancy. They have left the debate open with a note that the strong effect of emotions on different medical conditions cannot be ignored.

Rogers SN et al (1999)[44] have determined that edentulous patients after cancer treatment have more psychological disturbances and these may complicate compliance with prosthetic appliances.

Bocca M et al (1999)[45] in their study of 28 oral cancer patients found almost all subjects revealed the presence of anxiety symptoms and 60 percent of subjects were affected by minor depression.

In general the depression in a dental patient may be recognized by mood swings, unhappiness, loneliness, apathy, negative self- image, worthlessness and feeling of guilt. Many patients exhibit loss of interest in family, job and sex.

Continued feeling of tiredness, anorexia, insomnia and crying spells is seen in long-term depression. Malt UF et al (1997)[16] have mentioned 99 patients who felt that dental amalgam was causing multiple somatic symptoms. This study revealed that 62 percent of these suffered from chronic anxiety disorder and 47 percent suffered from major depression as compared to none in 93 of alternative group. This states how patient's reaction to something as harmless dental amalgam can get blamed for a problem that is patient's own.

SUMMARY

Many of the disorders mentioned in this chapter are closely related to depression. Either psychiatrist or the oral medicine specialist normally administers antidepressant therapy.

One major side effect of the antidepressant therapy is that it accentuates the Xerostomia in the depressed patient. Knowledge of the basic psychology is fundamental for effective and efficient practice in that it helps the doctor in management of both. The fear that most patients have, as well as this varied group of oral disorders result from the interaction of soma and the psyche. A knowledgeable dentist a should be able to recognize and refer the selected cases to psychiatrist and use the services of the medical social workers in long term follow up and total health care of his patient.

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FURTHER READING

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Bailoor DN, Nagesh KS

33 Occupational Hazards in Dentistry

INTRODUCTION

The study of occupational medicine is a highly specialized branch, which deals with risk of particular vocations. Dentistry is peculiar among the various health occupations, in that it makes both artistic and scientific demands on the practitioner. The dentist comes in close contact with patients for long time thus falling prey to infections. Technical advances make new dangers like radiation risk, electric shocks, hearing impairment and eye problems due to lasers, a reality.

HAZARDS

The practice of dental surgery is not as hazardous as air line pilot or police man's job but still relaxing ones vigil over the occupational hazards aspect can prove disastrous as we shall proceed to show in this chapter. Following are the major areas—

- Infectious diseases; RSI and orthopedic problems; noise and visual disturbances;
- Dental materials risk, allergy and equipment; radiation hygiene problem; stress and professional burnout; medicolegal problems; biowaste and problem with its disposal.

INFECTIOUS DISEASES

Dental surgeons run a high risk to different infectious agents—bacterial viral and fungal. In India, tuberculosis and hepatitis B appears to be a very real danger and AIDS has begun to spread like wild fire.

Methods of prevention of cross infection

- Identification of high-risk patients
- Highest possible standards of personal hygiene
- Disposable gloves, mouth masks, syringes, needles and zero-powered glasses
- Bactericidal chemicals like chlorhexidine, sodium hypochlorite, etc.
- Vaccination against hepatitis—B (using recombinant DNA technology) Engerix B®

Schatz et al[1] have determined the hepatitis B to be the prime infectious occupational hazard. In their careers at least one in six will become infected and in surgeries as many as 50 percent may contact the virus.

In case of practitioner gets contact of such patients it is advisable to seek physicians care. Dentists also have an important job of educating patients against the risk of AIDS in the community. He must follow all the recommendations of OSHA (occupational safety and health administration), which appear in the journals national and international from time to time.

The following patients belong to high-risk groups:

a. Male homosexuals and bisexuals.
b. Intravenous drug abusers.
c. Hemophiliacs who have received multiple blood transfusions.
d. People migrated from high-risk countries like those of South-east Asia Thailand, Myanmar, Indonesia, Korea etc., Sub-Saharan, African countries are another high-risk areas.
e. Promiscuous sexual behavior with any of the above groups.

Longer the dentist is in practice greater is the risk that he will get exposed to either hepatitis B or any related viral infection as per the study of Panis B et al (1986).[8]

Evidence regarding saliva being a carrier for HBV infection is being debated but it stands to reason that the bleeding from infected gums when mixed with saliva will be able to transmit the infection. The risk from hepatitis B can be summarized as:

i. Chronic carrier state is common in this disease
ii. The viruses survive well outside living bodies for long times and are resistant to routine disinfections
iii. Very small amounts of body fluids can transmit this disease
iv. Infection can lead to active hepatitis, liver cirrhosis and even hepatoma killing the patient.
v. With advent of newer strains like C,D,E,F,G,H etc and the hepatitis B itself mutating rapidly that we cannot rest smug that we have been vaccinated. Constant vigilance against cross infection should be the goal.

In India any patient who has confirmed tuberculosis needs to undergo ELISA 1+2 testing to rule out HIV infection. The port cities of Mumbai, Goa, Mangalore, Chennai, and Calcutta are hot beds of spread of AIDS. If any patients show oral thrush, advanced periodontitis now known as NUP (Necrotising Ulcerative Periodontitis) or non-healing ulcers and purpuric spots etc. almost confirms the immunodeficient status of such a patient. A completely disposable diagnostic kit and quick referral to specialized centers of HIV treatment is indicated. Most of the research shows that the occupational risk for HIV for dentists is extremely low and this has been confirmed many studies classical amongst them is N'Galy B et al (1988)[9].

HSV—Herpes Simplex Virus infection is one of the commonest to affect the dental staff and dentist occupationally. It was found by Brooks SL et al [10] (1981) that 40 percent of dental students and staff are non-immune to HSV in an American survey and are at risk. Even though no Indian studies in this respect were readily available the figures of non-exposure in India would be very low indeed. Some simple points to remember for purpose of cross infection control are as follows:

1. Use 2 percent chlorhexidine prior to all dental care in patients to reduce local infection.
2. Use latest autoclaves, disposables and disinfection procedures.
3. In the clinics do not wear street clothes, always use clinical attire that is of tieback variety and changed everyday.
4. Headgear or tying hair back with sterilized cap is always a good idea.
5. Wearing protective eyeglasses is a good practice.
6. Use of tray systems minimizes the working area exposed to infection and all areas to be swabbed by sodium hypochlorite 1 percent after industrial soap spray.
7. Rubber dam must be routinely used for isolating teeth or quadrants from saliva and spray spread of infection.

POSTURAL, ORTHOPEDIC PROBLEMS AND RSI

Pollack R (1996)[17] mentions that the dentists tendency is to adapt awkward physical postures to access the oral cavity. This causes different work-related musculoskeletal disorders. The science of Ergonomics allows us to design clinic ambiance in such a manner that dentists can sit straight backed on a correct height clinical stool and patient rests comfortably in open mouth position.

Bramson JB (1998)[31] defines Ergonomics as the science or study of workers and their adjustment or adaptation to the working environment or working conditions. He mentions that researchers used two human tech analysis tools.

1. Baseline risk identification of ergonomic factors (BRIEF)

2. Ergonomic assessment survey (EASY).

FIGURE 33.1: Showing wrong posture and bending over the patient can cause the low back pain and cervical spine problems (Prasanna K, Bailoor DN 2004 Yenepoya Dental College and Hospital, Mangalore)

These two analysis instruments designed to identify risk factors associated with various human components of work. The use of electromyography and goniometry complements the data analysis obtained from the above tools.

Fish DR and Morris-Allen DM (1998)[27] surveyed more than a 1000 dentists in Nebraska and found that the two commonest ailments affecting the musculoskeletal tissues were carpal tunnel syndrome and low back pain. Twenty nine percentof dentists reported symptoms of peripheral neuropathy in the upper limbs or neck (Fig. 33.2).

Augustson TE and Morken T (1996)[28] interviewed 329 employees in the Public Dental Service in Norway and found that 81 percent had some form of musculo-skeletal discomfort in the last 12 months. Low back pain was noted in 49 percent, neck pain in 47 percent and shoulder discomfort in 45 percent of the respondents. Neck discomfort became worse with age. Those dentists who participated regularly in sports had considerably less musculo-skeletal problems.

Papageorgiou AC et al (1997)[29] found that people dissatisfied with work are more likely to report low back pain.

Low back area and legs are two points where dental practitioner strains himself during stand up dentistry and bending often to see upper teeth and palatal areas (Fig. 33.1). In India many dentists work very hard for more than 8 hours without rest.

This may cause:

- Cervical spondylosis
- Low back pain
- Varicose veins
- Knee and ankle joint-osteoarthritis
- Rarely scoliosis and other vertebral column problems

Repetitive stress injuries (RSI) are becoming more common in dentists with increased use of computers,

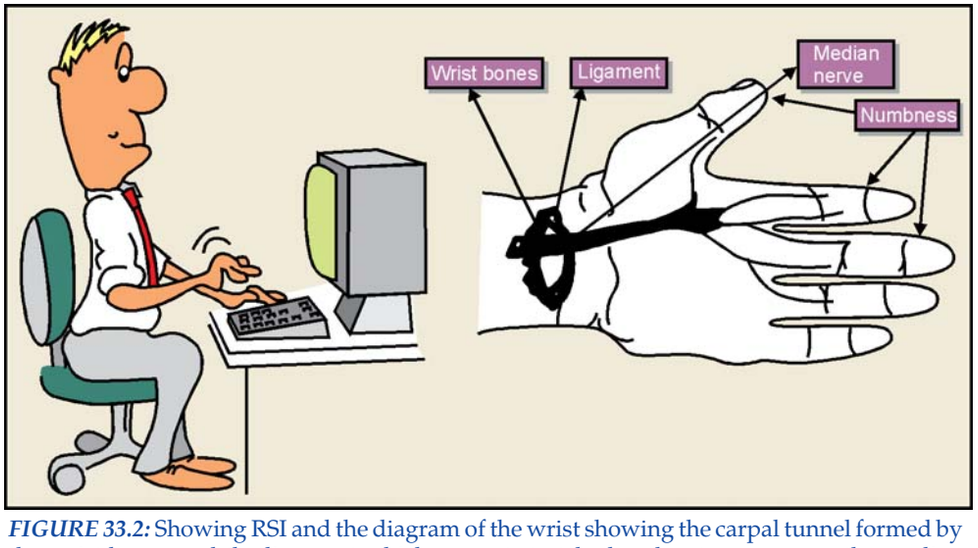

FIGURE 33.2: Showing RSI and the diagram of the wrist showing the carpal tunnel formed by the wrist bones and the ligament which can get stretched and put pressure on the median nerve resulting in weakness and numbness in the thumb, forefinger and the middle finger (Prasanna K, Bailoor DN 2004 Yenepoya Dental College and Hospital, Mangalore)

keyboarding and track ball and mouse use for prolonged periods. Many practitioners in India still use manual scaling and this can add to the stress on the wrist and fore arm region.

The nerves in the carpal tunnel get compressed due to overwork and thickening which leads to their inflammation. Pain and paresthesia in wrist and radial side of the hand, muscle weakness and abnormal nerve conduction are the common signs and symptoms of CTS.[34]

People of risk for CTS are luggage handlers, sonography technologist, secretaries, sales persons, crane operators, postal sorters, digital radiography specialists and journalists.

Some sportsmen like golf, bowlers in cricket and tennis players all may be at increased risk.

In rare cases children have been reported with something similar termed as CTS, which appears due to, inherited problems in metabolism. Van Meir N et al [35] have reviewed 163 cases in addition to 52 already published. The most common etiology was lysosomal storage disease: mucopolysaccharidoses (MPS) and mucolipidoses (ML).

Today children are playing more computer games and usage of digital media is universal in the classroom. In the coming years we can see an epidemic of these disorders if proper education of risks of computing is not made.

RSI is manifested due to:

1. Wrong posture in computer keyboarding
2. Over work
3. B- complex deficiency

RSI has been diagnosed by the occupational therapists as carpal tunnel syndrome, which involves median nerve, and the cubital tunnel syndrome, which involves the ulnar nerve.

These two are the most common compressive neuropathies. Commonly used diagnostic tests are:

A. Strength sensation and provocative testing
B. Grip strength measured with dynamometer
C. Two-point discriminative test
D. Elbow flexion test
E. MRI

It has been hypothesized that idiopathic carpal tunnel syndrome (CTS) is a manifestation of vitamin B6 deficiency. Aufiero E et al[33] mention that it appears reasonable to recommend vitamin B6 supplementation to people with CTS because B6 supplementation addresses an unrecognized peripheral neuropathy.

Following treatment is recommended for this neuropathy:

- Rest
- Workplace task modification
- Vitamin B-complex supplementation
- Nerve and tendon gliding exercises
- Steroid injection
- Wrist splinting
- Open wrist decompression
- Endoscopic carpal tunnel surgery

Best way to prevent postural problem is:

1. Space out professional work with rest
2. Use an assistant with sit down dentistry
3. Use ergonomically designed chairs and tables
4. In case of warning signs of persistent pain in any joints rest and orthopedic consultation must not be delayed.
5. Use of Yoga for posture correction and relaxation must be considered.

NOISE POLLUTION AND VISUAL DISTURBANCES

Use of high-speed turbines, faulty air conditioning can contribute to noise pollution in the clinics and cause hearing impairment. Ultraviolet light curing systems, computers and lasers can adversely affect the eyes and cause visual disturbances in the careless dentist. Manufacturers recommendation should be followed meticulously, safety glasses of different lasers and UV lights should not be interchanged. Using color monitors in clinic, computer field is not sufficiently illuminated operator may suffer from nodular headaches and loss of visual acuity. The ratio between general lighting to that of operating field should be 1:4 (Fig. 33.3).

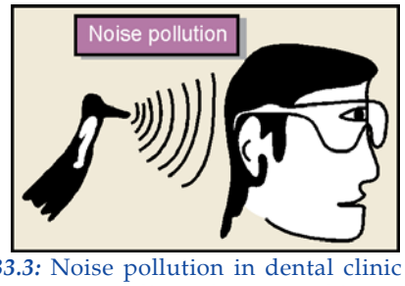

FIGURE 33.3: Noise pollution in dental clinic and dental laboratory can cause auditory damage and increased levels of stress (Bailoor DN, Nagesh KS 2004)

RISK RELATED TO THE DENTAL MATERIAL

Drugs and Equipments

Mercury Toxicity and Allergy

The greatest risk from mercury is from vapor in the air, handling of mercury specially through the region of the nails. The maximum level of exposure considered to be safe is 50 microgms/cc of air. Other dangers from mercury appear to be due to hypersensitivity.

Nixon and Smith (1964)[3] have described a dentist and his assistants suffering from profuse salivation, tremors of hand and insomnia and irritability. This was later traced to mercury poisoning.

Cook and Yates (1969)[4] have recorded a death of 42 years old dental surgery assistant who on autopsy revealed kidney failure due to mercury intoxication. All these examples throw light on the insidious and painless poisoning that may result from careless manipulation of mercury.

Bailoor DN (1991)[5] mentioned following steps to minimize the hazards ? Use automatic triturators ? ventilate your surgery with exhaust fans? Spilled mercury should be collected with gloved hands and stored under water? Spillage in inaccessible areas should be treated with flowers of sulfur.

Parmeshwaran et al (1993)[2] have graphically mentioned causes of mercury spillage and also highlighted the methods of reducing mercury hazards in the dental clinic.

Ritchie KA et al (1995)[25] performed a pilot study to analyze the impact of low-level exposure to mercury on the general health of dental surgeons. A computerized analysis was made of psychomotor responses and memory recall. Statistical analysis showed that the older dentists had slightly higher concentrations of urinary mercury but all within occupational limits. Memory recall also was affected in older dentists. It is important to differentiate natural memory loss due to ageing and this sub clinical exposure to mercury. Larger studies now been conducted will definitely throw further light on this matter.

Kostyniak PJ (1998)[24] has explicitly highlighted occupational impact of mercury vapor and confirmed safety protocols in dental clinics.

Osborne JW and Albino JE (1999)[26] reviewed the psychological and medical effects of mercury intake from dental amalgam and gave a status report. They mention that mercury is safe for use in dental amalgam since the amount released in the mouth is insufficient to cause any health problem. Removal of existing mercury fillings if attempted without proper suction and rubber dam would result in transient increase of serum mercury level. They also found that 89 percent of the patients with self-reported "amalgam illness" had psychogenic disorders, whereas only 6 percent of the matched-pair manifested symptoms of these psychological disorders. They opine that most of the so- called amalgam related medical problems are clearly psychological.

Chadwick RG et al (1994)[20] studied the combined affects of blue light of the curing units in order to determine whether they pose a dermatological hazard. They recommend eye protection and that gloves be worn routinely during working with such units. This will protect not only against UVA but also the catalysts and chemicals used.

Chowanadisai S (2000)[30] have reviewed the work place problems and said that modern dentistry has been described as least hazardous of all occupations but the exposure to new dental materials and infectious diseases may change this picture.

Dermatosis, eye problems, respiratory and systemic problems and musculoskeletal problems. Along with these problems glove dermatosis and latex allergy were also reported in dental personnel.

In injuries percutaneous injuries are reported in majority of the cases. A recent study of dentists found a high prevalence of musculoskeletal problems with reported backache and headache.

The common instruments that caused percutaneous injuries are hypodermic needles, hand scalers, burs, endodontic file, explorers, exodontic elevators and matrix bands.

Majority of allergies caused by were due to latex gloves, paper masks, acrylic powder, amalgum, vitalium dust, plastic eye glasses, stainless steel crowns.

The eye problems were noticed more in female dentists than in males. These include short sightedness, unclear sight, eye irritation, eye fatigue. Greater use of goggles and

eye shields has reduced the problem. The hearing problems were more in the previous years due to the high noise level of dental equipment. Now a days the noise level has falled below the standard bench mark level due to which the risk of hearing loss is minimum. Continuing education in sensitizing the dental personnel to the work related risks will go a long way in decreasing these problems.

Respiratory problems like pneumoconiosis can occur due to inhalation of methylemethacrylate vapors.

Anesthetic hazards: Halothane causes liver damage and nitrous oxide depression of vitamin B 12 absorption. Female staff if pregnant should avoid inhaling both of these which are established as teratogenic agents. Rowland AS et al [7] clearly mention that dental assistants exposed to high levels of nitrous oxide may impair women's fertility. In 1977, the National Institute for Occupational Safety and Health recommended an exposure standard of 25 ppm but the OSHA—Occupational Safety and Health Administration has never made the standard mandatory. Exposure levels are 100 ppm in offices with scavenging equipment and 1000 ppm in those without such devices.

Ineffective Drugs in Clinical Practice

It could be due to fake drugs. Another emerging problem is the factor of spurious drugs in the Indian market. The newspapers are flooded with reports about the fact that one-third of the drugs in some states may be spurious or substandard. The drug controllers of most states are trying to do their best, however, due to lax implementation of laws and the no real teeth to the punishment process even if some person is implicated in such cases results in the very low conviction rates and frustration in the enforcement authorities. Not enough staff seems to be problems besetting most state government departments.

The spurious drug industry is becoming well established in India. According to World Health Organisation's (WHO) 2001 statistics, 35 per cent of the world's spurious drugs are produced in India, followed by Nigeria at 23 percent. By all accounts the magnitude of this problem would have only increased in the last two years.[32]

Radiation Hygiene

Following proper safety measures in working with dental X-ray units is termed as radiation hygiene. The biological effects of radiation are broadly classified as somatic and genetic.

The somatic effects for the dental patients are negligible but the genetic effects thresholds are yet to be assessed for absolute safety. All the dental surgeons should remember that radiation effects are cumulative and that this insidious damage is totally painless.

Svenson B et al (1996)[21]based their study on 2000 mailed letters to Swedish dentists and found that there was a significant relationship between years in practice and higher training in oral radiology to reduce in the risks in dental clinic. Most dentists took their radiation risks very seriously.

Ansari IH (1997)[22] has cautioned about indiscriminate use of panoramic radiography for patients seeking complete dentures. He cautions that history and clinical examination should dictate whether radiograph is required and it should not become a administrative procedure. .

Following are the recommendations for safety of the practitioner.

- Buying of standard radiographic equipment, which rigidly follows the NCRP and ISI recommendations.
- Well collimated and filtered beam of at least 1.5mm of aluminum filtration should be available.
- Use of lead barriers between the dental surgeon and the X-ray machine is mandatory. Lead should be at least 2 mm thick. The lead should extend at least 12 inches below the floor level (Fig. 33.4).

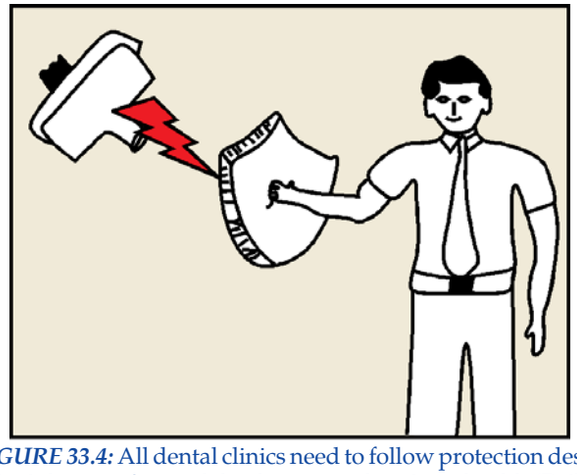

FIGURE 33.4: All dental clinics need to follow protection design principles to safeguard the doctors and patients health (Bailoor DN, 2004)

- Special Conch shell designs are recommended for the X-ray departments.
- The walls during construction use a special barium plaster which absorb the scattered radiation.
- Lead aprons should be routinely used for all patients, and all children special thyroid shield should be used.
- Use of fast films i.e. Ekta speed to lower exposure times.
- Dental surgeons must use a film badge service provided by the BARC Bombay for personnel monitoring.

Further information regarding the monitoring may be had from Chief Radiation Officer, BARC Division of Radiation Protection, Personnel Monitoring Section, Trombay, Mumbai.

The entire planning should be done such that ALARA principle is followed at all times, i.e. as low as reasonably achievable.

Frazier LM and Jones TL (2000)[23] commented about managing patients with concerns about workplace reproductive hazards and found in their series of 51 cases that only one man attended the occupational health clinic compared to 50 women. This means that the paternal contribution to birth defects is still highly underestimated. Women working in this series were occupationally involved with ionizing radiation, biological hazards, electromagnetic fields, and ultraviolet light. Another finding was that the mean gestational age of the fetus was 10.9 weeks in this study, which meant that most women consulted too late. A lot of stress needs to be given on educating the men and women in work places like dental clinics.

Stress and Burnout

Professional burnout, a long-term consequence of occupational stress, is considered to be a factor that explains a substantial proportion of incapacity for work. Burnout is defined as emotional exhaustion.

Bourassa M and Baylard JF (1994)[12] have highlighted that interpersonal relationships involving patients and/or office personnel was probably one of the important areas for stress generation. Dentists belonging to the older age groups managed stress much better than their younger counterparts.

Humphris G et al (1997)[15] studied 52 junior hospital dentists using the Maslach Burnout Inventory and found that 10 percent of respondents were suffering burnout. Depersonalization (a result of extended and demanding contacts with patients) was significantly ($P < 0.05$) greater in restorative and oral surgery specialties.

FIGURE 33.5: Proper organization and time management will lead to reduction of stress in dental office environment (Bailoor DN 2004)

Freedman G et al (1997)[11] have stressed that selection of suitable equipment and proper posture during working preferably in sitting down position will go a long way in reducing stress.

Today doctors are expected to keep a lot of paper work like the Form 3C which is daily record of the patients and their payment, record of expense and income, bank loan repayment records, insurance of the premises and equipment, and practice indemnity insurance. In addition the EPA Act 1998 mandates that clinics which treat more than 1000 patients a month should keep record of the biowaste disposal (Fig. 33.5).

Keeping all the records, facing the patients, the hard work of precision and the over expectations all build up to a tremendous stress which can cause numerous health problems.

Wilson RF et al (1998)[13] performed postal survey amongst 1007 general dental practitioners and found that improper time management was one of the most important stressors.

Gorter RC et al (1998)[14] found that among 709 Dutch dentists a lack of career prospects appeared to be the stress factor most strongly related to burnout.

Kulich KR et al (1998)[18] conducted their survey on 64 Swedish dentists and found that importance of interpersonal skills, stress tolerance and administrative skills was found very useful by experienced clinicians. They also noted that currently none of these were emphasized in the universities today.

Gorter RC et al (1999)[19] found in their study that 13 percent of middle aged dentists suffered from high levels of burnout. In their example they found that male dentists in their forties were most likely to suffer from stress related problems.

Mazey KA (1994)[16] suggests 5 coping strategies for stress reduction (i) seeking information, (ii) taking direct action, (iii) inhibiting action, (iv) engaging intrapsychic efforts (v) calling on others.

The following factors are said to contribute to psychological stress in practitioners.

1. A practitioner who is too perfectionist.
2. One who is very highly ambitious who may be seldom satisfied?
3. Type A personality one who wants to do everything today, who talks loudly, and quickly and feels constantly the pressure to excel (Fig. 33.6).

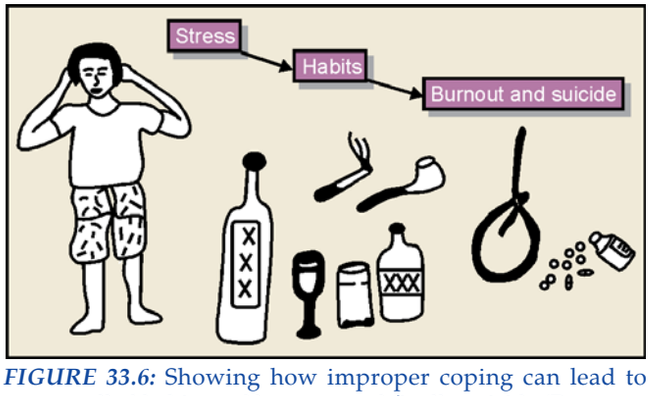

FIGURE 33.6: Showing how improper coping can lead to uncontrolled habits and burnout and finally suicide (Prasanna K, Bailoor DN 2004 Yenepoya Dental College and Hospital, Mangalore)

Such doctors frequently fall prey to disillusionment leading to grave tobacco and alcohol abuse. Some even take the readily available drugs. All this leads to poor co-ordination, clouded thinking and mistakes. This brings professional shame and further enslavement to habits. Finally to depression and even withdrawal from work as well as family attachments. This untreated can lead to burnout a process where person is socially and professionally compromised to the point of requiring psychiatric treatment.

Schubot and Green [6] have proposed the following strategies for coping:

- Developing awareness of the problem
- Responsibility of taking some action
- Clarifying ones thoughts and solutions
- Engaging in coping behavior

This coping behavior comes in various forms—

1. Taking frequent vacations at least bi-annually.
2. Developing hobbies like gardening, tennis, swimming etc. which make physical demands. This in itself helps in combating work stress and depression.
3. Having strong family ties, and compulsory Sunday off makes one recuperate.

In case you see such behavior in close professional colleague you must make it point to get involved and help him, and if required advise a psychiatrist's opinion.

MEDICOLEGAL PROBLEMS

In India, dental practice has been included in the purview of the Consumer Protection Act. According to this, the faults on the part of the practitioner may be classified as:

- **Fault of commission**—The act of giving wrong or unnecessary treatment which may aggravate the disease, for example in case of diabetic patient, extracting the tooth without assessing the serum glucose level and therapy causing cellulitis and even death if diabetes is uncontrolled.
- **Fault of omission**—The act of withholding a correct treatment which may aggravate or kill the patient, for example if patient has an anaphylactic attack—then not giving him adrenalin 1:1000 1cc followed up by Injection Betnesol® (Steroid) can be fatal to the patient.

The dental surgeon can minimize medicolegal risks in his practice by:

1. Maintaining correct dental records.

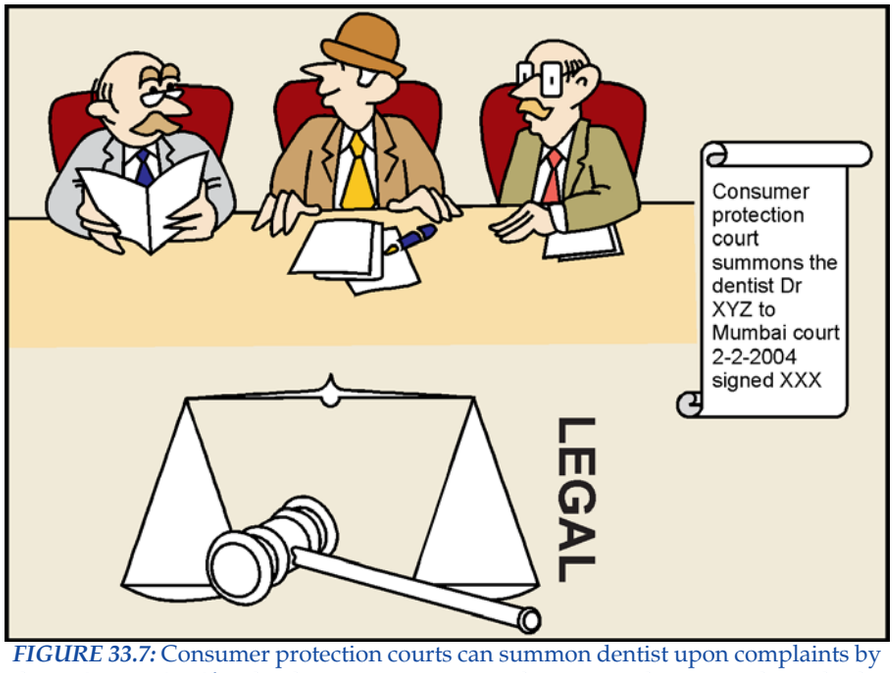

FIGURE 33.7: Consumer protection courts can summon dentist upon complaints by the patients to justify why the treatment was not done as per the accepted standards. Another dentist or professional who is specialist in the area is expected to testify that procedures were not followed. There are local, state and national protection bodies (Prasanna K, Bailoor DN 2004 Yenepoya Dental College and Hospital, Mangalore)

2. Obtaining informed consent from patient/parents in case of children.
3. By attending conferences, workshops and continuing dental education programs to keep abreast of the latest.
4. Lastly the dental surgeon can cover himself using Professional Indemnity Insurance in event of a malpractice suit.

Other possible areas could be:

a. Professional misconduct.
b. Breach in Government service rules for employed doctors.
c. Failure to comply with OSHA occupational safety and health act rules to protect the staff working in the clinics (Fig. 33.7).

BIO-WASTE DISPOSAL AND RELATED PROBLEMS

Biomedical waste is the waste that is generated by the hospitals, nursing homes or clinics during the diagnosis, treatment or immunization of human beings. The animals being processed as food or in research activities pertaining thereto will also lead to waste which will be included in this group.

The institutions that generate such waste have a responsibility to see that this waste does not cause pollution and pose to be a health hazard.

The organization should approach the Pollution Control Board of the region to obtain a proper authorization for the way the waste is being disposed.

The waste needs to be segregated in six categories as mentioned by the rules and in general it's a requirement to separate the sharps from non-sharps and infected and non-infected material. Please check the requirement with your local Pollution Control Board and your state authorities and get appropriate authorization for your dental clinic.

A lot of international agencies are working in form of NGO's like Shristi working in Bangalore with Health Care Waste Management Cell of M.S. Ramaiah Medical College. They are set to lend a helping hand to the World Health Organisation to sensitize professionals against the legal obligations of the waste management.

Bhumata charitable trust and United States-Asia Environmental Partnership (US-AEP) are doing yeoman

Following Central Government of India notification has been issued which the practicing doctors should be familiar with.

This notification issued under powers conferred by Sections 6, 8 and 25 of Environment Protection Act, 1986.

RULE 2: The rules apply to all persons who generate, collect, receive, store, transport,treat, dispose or handle biomedical waste in any form.

RULE 3: Biomedical Waste means any waste, which is generated during the diagnosis, treatment or immunization of human beings or animals or in research activities pertaining thereto or in production or testing of biological, and including categories mentioned in Schedule I; Biomedical Waste treatment Facility means any facility wherein treatment, disposal of biomedical waste or process incidental to such treatment or disposal is carried out; Occupier in relation to any institution generating biomedical waste, which includes a hospital, nursing home, clinic, dispensary, veterinary institution, animal house, pathological laboratory, blood bank by whatever name called means a person who has control over that institution and/or its premises; operation of biomedical waste facility means a person who owns or controls or operates a facility for the collection, reception, storage, transport, treatment, disposal or any other form of handling of biomedical waste.

RULE 4: It shall be the duty of every occupier of an institution generating biomedical waste which includes a hospital,nursing home, clinic, dispensary, veterinary institution, animal house, pathological laboratory, blood bank, by whatever name called, to take all steps to ensure that such waste is handled without any adverse effect to human and the environment.

RULE 5: Biomedical waste shall be treated and disposed of in accordance with schedule I and in compliance with standards prescribed in schedule V… every occupier.. shall set up …. Requisite biomedical waste treatment facilities … or, ensure requisite treatment of waste at a common waste treatment facility or any other waste treatment facility.

RULE 6: Biomedical waste shall not be mixed with other wastes …shall be segregated into containers/bags at the point of generation in accordance with schedule II prior to its storage, transportation, treatment and disposal. The containers shall be labeled according to schedule III. If a container is transported … to any waste treatment facility outside the premises, the container shall … also carry information prescribed in schedule IV … Untreated biomedical waste shall be transported only in such vehicle as may be authorized … No untreated biomedical waste shall be kept stored beyond a period of 48 hours … (conditional)

RULE 7: The Government of every state … shall establish a prescribed authority with such members … for granting authorization and implementing these rules … appointed within one month of the coming into force of these rules … The prescribed authority shall on receipt of Form I make such inquiry as it deems fit and if it is satisfied that the applicant possesses the necessary capacity to handle biomedical waste in accordance with these rules grant or renew an authorization … An authorization shall be granted for a period of three years, including an initial trial period of one year from the date of issue. All such subsequent authorization shall be for a period of three years. A provisional authorization … for the trial period (to be issued) to enable the occupier/ operator to demonstrate the capacity of the facility ….

RULE 8: Every operator of biomedical facility … (and) …. Every occupier of an institution generating, collecting, receiving, treating, disposing and/or handling biomedical waste in any other manner, except such occupier of clinics, dispensaries, pathological laboratories, blood banks providing treatment to less than 1000 patients per month …. shall make an application in Form I to the prescribed authority for grant of authorization. Every application … shall be accompanied by a fee as may be prescribed by the government of the state ….

RULE 9: The Government of every state … shall constitute an advisory committee, will include experts in the field of medical and health, animal husbandry and veterinary sciences, environmental management, municipal administration and any other related department or organization including non-governmental organizations. The State Pollution Control Board/ Pollution Control Committee shall be represented …. The Advisory Committee shall advice the government of the state … and the prescribed Authority about matters related to the implementation of these rules.

RULE 10: Every occupier/operator shall submit an annual report … by 31 st January every year, to include information about the categories and quantities of biomedical wastes handled during the preceding year.

RULE 11: Every authorized person shall maintain records related to generation, collection, reception, storage, transportation, treatment, disposal and/or any form of handling of biomedical waste in accordance … and be subject to inspection and verification … at any time.

RULE 12: When any accident occurs at any institution or facility or any other site where biomedical waste is handled or during transportation of such waste, the authorized person … shall report … forthwith.

All over India private agencies have been given the contract and empowered to collect this bioactive waste and dispose it in a rational manner. The three given below are representative of such organizations.

1. Bio-Care Technological Services,
   55, Railway Road, Samaipur Industrial Area, Delhi-42
   Phone No. 27898011, 27898033, 27866142
2. Synergy Waste Management Company,
   5/2-B, Asaf Ali Road, New Delhi - 110 002.,
   Phone No. 23222522, 23222622, 25968318
3. Maridi Ecotherm Systems Ltd.,
   Jer Mahal, Ist Floor,
   Dhobi Talao, P.O.Box: 2352
   Mumbai - 400 002.
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   Fax: 0091-22-22075501.

service in the Pune region of Maharashtra in creating training sessions for doctors and hospital managers in specialty of waste management.

Vatavaran, a NGO working on socio-environmental issues, carried out a study on hospital waste issue. The group studied the collection, segregation and disposal of hospital waste in the capital and warned "not all hospital waste" makes it to the incinerators. Often they find their way back to hospitals or more dangerously spill over into homes. This report by Bindu SP in Hindu Newspaper [36] has clearly highlighted that though laws exist and agencies flourish the implementation and protection of general public's health is not attained at all. It is up to all of us dentists, doctors and health care professionals to see that we all assist the authorities in containing the pollution and the health hazard posed by biological wastes.

SUMMARY

We may summarize that this knowledge of occupational hazards will help the practicing dentist by helping him to prevent these risks, and as the old adage goes "Prevention is better than cure". In occupational medicine these risks can be reduced by:

1. Primary prevention, i.e. making the newly trained doctors aware of these problems and promoting ergonomically designed dental clinics.
2. Secondary prevention involves regular screening of professionals for early evidence of occupational diseases. Local dental associations or government liasoning bodies like State Dental Councils can do this work.
3. Tertiary prevention techniques are those that utilize regular screening for the practitioners who have developed some of the above-mentioned problems and then take corrective action and suggest life style modifications.

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34 Lasers in Dentistry

INTRODUCTION

It was the genius of Einstein, which proposed the theory of the spontaneous and stimulated emission of radiation, which was published in 1916. Number of pioneers worked in this area and finally it was to, Townes, Basov, and Prokhorov the Nobel Prize for development of laser was awarded, in 1964. This first laser was a Pulsed Ruby Laser. A lot of initial work in dentistry was done using the Ruby Laser. Ruby Laser had very limited success in dental treatment due to its high heat effects and thermal related damage to the dental tissues.

PHYSICS OF LASER

Maiman TH may be called as the Father of Lasers. In 1960 he worked with the Hughes Aircraft Corporation and developed what was termed as the MASER. This was an acronym for microwave amplification by stimulated emission of radiation. Recently the acronym LASER means Light Amplification by Stimulated Emission of Radiation.

The laser light has following three characteristics which differentiates it from the normal light.

a. Monochromatism
b. Coherency
c. Collimation

Monochromatism is the single color being used, Coherency is the capacity of these photons to be in sync with each other so that their energy is amplified, and Collimation is the fact that the beam is well focused and does not spread out like ordinary light.

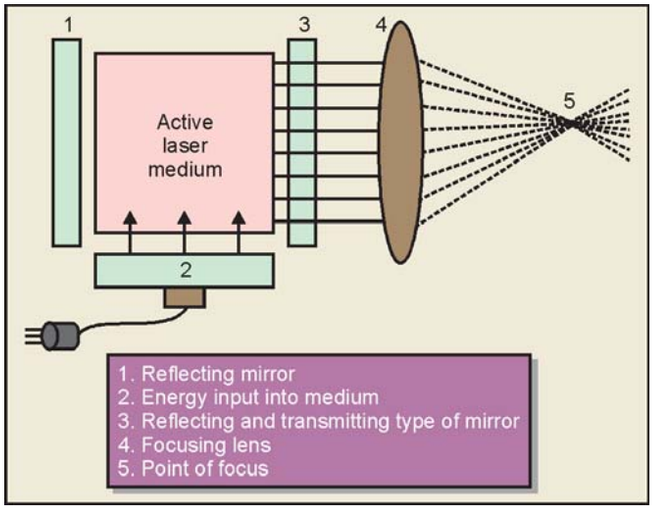

FIGURE 34.1: Showing basic principle of laser generation from energy input to internal reflection in the lasing medium to the focusing of the laser at a fine point

The physics of production of the lasers is essentially out of scope of this text, but in the coming lines we shall focus on the various clinical implications and safety procedures (Fig. 34.1).

The delivery of the laser energy to the dental tissues is usually accomplished using the—

1. A special hand piece type contraption, which allows the internal reflection and gives the high pulse of energy to the oral mucosa through non-contact mode.
2. Glass fiber optic cable of sizes ranging from 200- 1000 microns that is located in a sheath and used as contact on to the lesion.

The light energy is delivered to the tissues in one of the three modes—The continuous mode, the pulsed mode and free running pulsed mode. Basically either the energy is directed in an un-interrupted flow of photons or in blinks of light. The beam can be focused using a lens and when the laser is used exactly at its focus it is termed as in cutting mode and the tissue cut is sharp, deep and incisive. When however the laser is used in slightly out of focus mode then it is called as the ablative therapy. In this a broader band of tissue is coagulated superficially. It is obvious that these modes will find differing clinical applications (Fig. 34.2).

Interaction with Biologic Tissue-Hard Tissues-Dental Pulp

The laser and the target tissues interact in the following four physical ways-

1. Reflection
2. Absorption
3. Transmission
4. Scatter

The photochemical and photoacoustic are the two other ways that the lasers affect the tissues. The photochemical properties have been used for what is now termed as the PUVA therapy.

SAFETY CONSIDERATIONS

- ***For the patient:*** It is best that the patient closes his eyes, and sterile saline pads are placed on his eyes and recommended eye safety wear put on.
- ***For the operator*** the best safety is use of equipment is good working condition, proper training in its use and the recommended eye wear in form of goggles. Under no conditions the eyewear of two manufacturers be interchanged or any kind of laxity practiced due to overconfidence. Other cross infection methods should be practiced as usual. A special printed label is available and should be placed outside the place where the lasers are in use so that the auxiliaries and other personnel are duly warned.

TYPES OF LASERS

Every year the changing technology is bringing forth new types of lasers. This is just a random sampling.

- Ruby laser
- Carbon dioxide laser
- YAG laser (yttrium-aluminum-garnet) Neodymium—Erbium—Holmium A variety of solid state lasers with a crystal rod composed of yttrium-aluminum- garnet which is doped with other elements. Depending on these elements the properties of laser that is emitted is determined.
- Diode solid state laser
- Others—ArF excimer, frequency doubled Alexandrite, frequency doubled Nd:YAG, and free electron lasers.

SOME CLINICAL APPLICATIONS

Diagnostic uses: Laser fluorescence for the caries detection

Laser Doppler flowmetry to monitor pulpal and gingival blood flow patterns.

Laser Doppler vibrometry—to measure tooth mobility

Holographic imaging using the laser has been established for measuring the soft tissue three-dimensional pictures, tooth movement and tooth mobility.

Biostimulation—Certain low energy lasers have been used for stimulation of wound healing, treatment of dentinal hypersensitivity and even treatment of Carpal tunnel syndrome by Smith CR et al (1995)[1].

Herpes labialis was treated by Parkins F et al [3] in a free running pulse mode using Nd:YAG lasers. They claim that the pain was considerably less and recurrence rate reduced.

Photodynamic therapy uses the lasers for the treatment of the malignancies and the pre-malignant lesions and conditions. Application of 5- aminolevulinic acid followed with gold-vapour laser is found to be effective treatment in premalignant and squamous cell carcinoma.

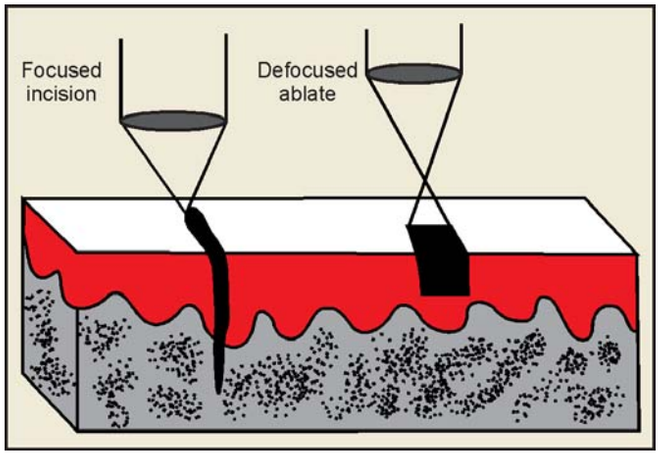

FIGURE 34.2: Showing the difference in cut mode and ablation mode of the laser. The depth of biological effect in focused mode is deeper than in ablation mode

Pediatric Dentistry

Soft tissue laser—CO_2 laser and Nd:YAG lasers are commonly used to cut the fibrous tissue over an late erupting permanent tooth, frenectomy or operculectomy overpartially erupted second molars. Colvard M and Kuo P (1991)[2] have demonstrated the use of managing the aphthous ulcers with Nd: YAG and found that the healing was quicker and uneventful.

Pulpectomy procedures are aided by laser debridment and the fiber optic system now is established in the endodontic literature for various endo procedures.

Er:YAG lasers have been cleared for all the ages for cavity cutting, and the first company to obtain the FDA clearance was the Premier Laser Systems® Irvine California. These lasers help in cavity cutting and contouring of the caries removed areas in such a way that they are sterilized and ready for many types of restorative materials. Low pulsed lasers also automatically etch the hard tissues.

Composite resin restorative materials can be cured using an Argon laser which has the 488 micron segment which matches exactly with the camphoroquoine, the photoactivator in many composite resins. This definitely improves the physical properties of the resins. High energy bleaching has been successfully reported for many adolescents specially in tetracycline stain studies and for other esthetic uses.

Maxillofacial surgery: The Carbon dioxide and the Er: YAG lasers are usually used. In focused mode they act as high powered light scalpels and in the de-focussed mode that is the ablative or vaporization mode it helps to superficially vaporize large lesions like leukoplakia, lichen planus, erythroplakia etc.

The usual advantages of sealing off of the blood vessels and sterilization of the surgical field results in superior postoperative healing.

Cosmetic laser surgery to remove skin wrinkles, nevi and other birth marks has been widely reported in the literature and is being tried with varied results in all major centers in India.

Arthroscopic laser surgery can be utilized to attempt fibrous tissue removals to relieve the fibrous ankylosis. Koslin MG and Martin JC (1992)[4] have used a fibre-optic tube system, which allows visualization and delivery of the pulsed laser energy at the precise intra articular point. They reported excellent postoperative results in the TMJ surgeries performed.

ADVANTAGES OF THE USE OF DENTAL LASERS IN DENTAL PRACTICE

- They cause reduction in the bacterial count and in some areas may sterilize the field as well. Therefore for patient at risk of bacteremia, lasers may be especially beneficial.
- Laser offers the ability to negotiate curves and folds in the oral cavity and depending upon the power setting and mode of delivery they can cut, vaporize or coagulate tissue.
- Laser seals the blood vessels. They offer a dry operating field and excellent visibility thus reducing the operating time.
- Laser seals the lymphatic vessels, which yields minimal postoperative swelling.
- With the use of laser pain is reduced to absent in 90 percent of the cases probably due to sealing of the nerve fibers and other 10 percent there will be pain of various intensity and duration.
- They cause less chance for mechanical trauma, minimal scarring and sutures are rarely needed.
- Enhanced patient acceptance due to minimal postoperative discomfort.

SUMMARY

This article is merely an introduction to the vast literature now available in the utilization of lasers as one of the tools in providing treatment for various lesions ranging from caries, gingival enlargement to major surgical explorations of hemagiomas and photodynamic therapies of premalignant and malignant lesions in the oral cavity. The likelyhood of the laser replacing the dreaded dental drill in looming on the horizon faster than we all expected.

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35 Clinical Pharmacology: Corticosteroids in Dentistry

INTRODUCTION

The optimal management of the dental patient receiving corticosteroid therapy is a growing dilemma. Consultation with the patient's physician or endocrinologist can maximize optimal dental care.

PHYSIOLOGY AND PHARMACOLOGY

Adrenal corticosteroids are necessary regulators of homeostatic life processes. These natural hormones include, glucocorticoids—hydrocortisone; the mineralocorticoids, aldosterone which affects water and electrolyte metabolism and the sex hormones; testosterone, estrogen and progesterone. Glucocorticoids secretion is primarily stimulated by pituitary adrenocorticotropic hormone (ACTH) and thus exogenous glucocorticoids suppress the hypothalamic—pituitary—adrenal axis (HPA).

The natural glucocorticoids, hydrocortisone is produced in the adrenal cortex from plasma cholesterol at a rate of 15 to 30 mg/day, in a daily rhythmic and pulsatile fashion[1]. The secretory rate is controlled by the HPA axis with the feedback inhibition (Fig. 35.1).

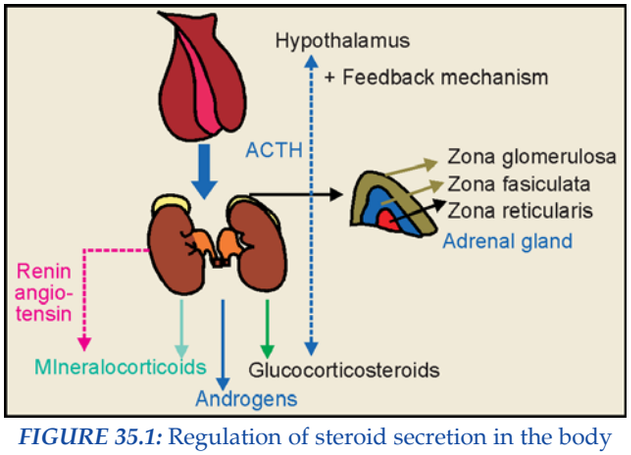

FIGURE 35.1: Regulation of steroid secretion in the body

MECHANISM OF ACTION

Actions of corticosteroids are mediated through the 'Lipocortin'. The corticosteroids enter the cell nucleus, which stimulates to produce the specific m-RNA for Lipocortin synthesis. The Lipocortin-mediated actions, which include,

- Inhibition of 'phospholipase' and thus reduction in the synthesis of Prostaglandin's- anti-inflammatory effect.
- Inhibits the production of 'Interleukine –II', which is secreted by the lymphocytes which in turn stimulates the proliferation of T-lymphocytes. These cells are responsible for the cell-mediated immunity.
- Inhibits the release and response of lymphokines

CLINICAL APPLICATION (Remembered as EROS)

1. Emergency therapy—In anaphylaxis and asthmatic attack
2. Replacement therapy—Adrenal insufficiency
3. Organ transplants procedures
4. Suppression of immune response—Immunologically-mediated disease.

Medical Indications[2]

- Adrenal cortical dysfunction—Addison's disease
- Cardiac dysfunction—Infective myocarditis
- Gastrointestinal dysfunction—Ulcerative colitis
- Hematologic disorder—Thrombocytopenic purpura
- Renal disorders—Glomerulonephritis
- Liver disorders— Hepatitis
- Allergy—Asthma, anaphylaxis
- Malignancy—Leukemia, lymphoma, myeloma
- Dermatological disorders—Psoriasis, keloids, pemphigus
- Collagen disorders—SLE, rheumatic fever, scleroderma
- Organ transplant— Kidney transplants
- Anti-inflammatory therapy—Trauma to CNS

Dental Indications[3]

- Pulp capping—Direct or indirect
- Endodontic sealer
- Aphthous ulcer
- Atopic
- Lichen planus
- Glossodynia
- Traumatic ulcer
- Oral submucous fibrosis
- Desqaumative gingivitis
- Oral erythema multiforme
- Oral pemphigus
- Desensitization of dentin
- Intra-articular injection to TMJ

CONTRAINDICATIONS (CAUTIONARY INDICATIONS)[4]

- Peptic ulcer
- Diabetes mellitus
- Hypertension
- Pregnancy
- Psychosis
- Tuberculosis
- Herpes infection
- Glaucoma
- Fungal infection
- Osteoporosis

ACTION OF CORTICOSTEROIDS[5]

1. *Carbohydrate metabolism*—Increase the blood sugar—Aggravates the pre-existing diabetes mellitus—Pre-diabetic patients can tend to become frankly diabetic.
2. *Protein metabolism*—Muscle wasting—Loss of bone matrix—Increase in capillary fragility—Inhibition of healing and fibrosis
3. *Fat metabolisms*—Moon facies and buffalo hump
4. *Mineral metabolism*—Sodium and water retention—Increased potassium ion- Edema formation—Increase in blood pressure
5. *Tissue inflammation*—Reduced
6. *Immunity*—Cell-mediated immunity is suppressed
7. *Mood*—High doses produce euphoria
8. *Anti-vitamin D action*—Absorption of calcium from gut decreased—Osteoporosis
9. *Adrenocortical suppression*—Growth of children arrested.

SIDE EFFECTS OF CORTICOSTEROIDS

As a dentist we should be aware of following side effects and look out for any initial manifestations of the same in individuals on steroid therapy.

1. *Altered response to physical stress*— Continuous usage of steroids for even a week leads to suppression of HPA suppression. The innate production of steroid is suppressed, making an individual vulnerable to stresses.
2. *Infection*—Long-term steroids can suppress the protective role of your immune system and increase the risk of infection.
3. Gastrointestinal ulcers or bleeding
4. Osteoporosis
5. Weight gain—Steroids affect metabolism and how body deposits fat. This can increase appetite, leading

to weight gain, and in particular lead to extra deposits of fat in abdomen leading to cushingoid habitus.

6. *Insomnia*
7. *Mood changes*—Doses over 30 milligrams per day, can affect moods. Some people can feel depressed, irritable or anxious.
8. *Fluid retention and elevated blood pressure*—As cortisone is involved in regulating the body's electrolyte balance, using these drugs can promote fluid retention and cause or worsen high blood pressure.
9. *Elevated blood sugar*
10. *Eye problems*—Steroids can cause cataracts or glaucoma or worsen these conditions if they are already present.
11. *Atherosclerosis (Hardening of the arteries*)—Steroids may increase the rate of development of atherosclerosis, which could increase risk of heart disease.
12. *Aseptic necrosis*—At higher doses, steroids can lead to aseptic necrosis of bone.

PREPARATIONS

- Betamethasone tab: 0.5 mg (Betnesol, Walacort, Celestone, Betnelan) ® Injection: 4 mg/ml (Betnesol)® Ointment:
- Dexamethasone tab: 0.5 mg (Decadron, Dexona)®
- Triamcinalone tab and ointments (Kenacort, Ledercort, Tricort, Kenalog)®
  Ointment: Kenalog in Orabase®
- Prednisolone; Betacortil, Wysalone

SOME CLINICAL SITUATIONS

- Hurtado Garc´ia JF et al (1997)[6] treated 47 patients of Bell's Palsy with initial 60 mg of prednisone® and followed up by oral steroids deflazacort® in a tapering regimen. Ninety five percent of the patients regained complete motor control. Authors recommend early intervention of Bell's palsy cases and state that age and medical complications like hypertension and diabetes are negative factors against healing.
- Ko JY et al (2000)[10] treated 30 patients suffering from facial palsy secondary to herpes zoster infection of the auriculotemporal region. Combination of parenteral acyclovir and oral prednisolone showed dramatic relief in most cases. Age and multiple nerve involvements were major negative prognosticating factors.
- Trovato C et al (1999)[7] have reported hypopharyngeal dysphagia as a complication of long term steroid treatment in 44 year old patient who was undergoing therapy for some collagen disease.
- MacPhail L (1997)[8] has implied that the most effective treatment for recurrent aphthous ulcers is the systemic or topical steroids and thalidomide. These agents are involved in the suppression or modulation of the immune system. The local steroids do not work effectively because in spite of vehicle like carboxymethyl cellulose they tend to wash of rather easily.
- Lu SY et al (1998)[9] followed up 41 patients with chronic oral ulcers in a 3-year open clinical trial. The patients had a clinical diagnosis ranging from oral lichen planus, erythema multiforme, and mucous membrane pemphigoid and pemphigus vulgaris. They were given levamisole 150 mg per day, prednisolone 15 mg/day and also local application of dexamethasone in orabase, Dexaltin®. In eight weeks of treatment all the patients in the series were found to be ulcer free. Authors recommend that addition of levamisole to the low doses of steroids can significantly improve the prognosis in these series.
- Chen HM [11] et al report from National Taiwan University, Taipei, Taiwan about a patient who suffered from facial cellulites caused by *Candida albicans*. This patient was an undiagnosed case of diabetes mellitus and who was suffering from oral submucus fibrosis OSF was treated with intralesional steroids (40 mg triamcinolone) biweekly and two months after the last injection the patient suffered from severe candidal cellulites. He had to be treated with amphotericin B intravenous 100 mg once a day for one week. Physician treated the patient for diabetes and the medical emergency was averted.

High school and college sportsmen abuse anabolic steroids to enhance their performance and it is reported widely in schools in the US.[12] The incidence of sports men using steroids in India is still not established but it is good

idea to check for signs of steroid abuse if the youngster has a pumped up demeanor. The team dentist can caution the sportsmen against using the drugs that will provide short-term benefits and completely ruin the health in the long run.

CONCLUSION

The judicious use of corticosteroids is both satisfying and lifesaving for the patient. A word of caution these powerful medications are double edged weapons, always weigh the risk versus benefit for the patient and keep in mind the long term compromise that may be precipitated.

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This chapter is not a replacement for professional dental training. Kindly verify the latest prescribing practices with your teachers and consultants prior to making real life decisions. Most values are indicative and have been checked against latest reliable sources, but the publishers and editors do not have any direct or indirect liability to the use or misuse of this prescribing information. Prior to prescribing any medication please check that they are from ethical drug manufacturers following sound quality control practices. Follow the manufacturers directions in most prescriptions and in case of new drugs please confirm side effects, safety in children and pregnancy with the nearby-approved University hospital specialists and legitimate internet sources.

—Editors

Shenai Prashanth, Chatra LK,
Bailoor DN

36 Applied Clinical Pharmacology

Antibiotics, Analgesics and Anxiolytics in Dental Practice

INTRODUCTION

Today the use of therapeutic drugs by the clinician requires thorough knowledge of molecular mechanism of action, pharmaco-therapeutics, toxic effect of that drug etc. Drugs are used in curative, suppressive and preventive way. The discovery of newer drugs have given the clinician widest range to choose from. Though numerous drugs are used in dentistry, this chapter shall limit it self to anti-microbial and analgesics which are commonly used in dentistry. As the role of psychosomatic factors in many oral diseases is becoming more evident, a brief discussion on anxiolytics and antidepressants also becomes relevant.

ANTIBIOTIC EFFECTIVENESS

It should be remembered that drugs are seldom the sole instruments of cure. The natural defense mechanism of the body performs the final elimination of the infection. The effectiveness of the given antibiotics depends on following factors.

Mechanism of Action

Antibiotics usually have two type of action.

1. ***Bacteriostatic:*** Acts primarily by arresting the bacterial growth, e.g. Sulphonides, Tetracycline, Chloramphenicol, Erythromycin
2. ***Bactericidal:*** Acts by killing the bacteria, e.g. Penicillin, Aminoglycosides, Cotrimoxazole, Ciprofloxacin, Isoniazid.

Some of the bacteriostatic drugs act as bactericidal at higher concentration. Bactericidal drugs tend to act faster and are not as dependent on exact dosage timing for their continued effectiveness.

Spectrum of Action

Antibiotics are often grouped as narrow, extended and broad-spectrum antibiotics.

Narrow spectrum antibiotics are most preferred as they cause less alteration of gut micro flora and few adverse affects, e.g. Penicillin G, Streptomycin, Erythromycin.

Extended spectrum antibiotic have some more wide range of activity, e.g. Ampicillin, Amoxycillin and Cephalosporins.

Broad-spectrum antibiotics are affective against a large variety of microorganisms, e.g. Tetracycline, Chloramphenicol.

Other Facts

That determine the antibiotic effectiveness. Development of resistance to an antibiotic might decrease the antibiotic effectiveness. There are many mechanisms by which bacteria develop resistance. It may produce beta-lactamase,

which inactivates penicillin and cephalosporins. Decreased binding to bacterial ribosomes may cause resistance to erythromycin and clindamycin.

Antibiotic penetration into the alveolar bone is crucial in many odontogenic infections. Clindamycin and cephalosporins produce highest alveolar bone concentration in contrast to penicillin. Inadequate drainage, foreign body reaction and duration may alter the antibiotic effectiveness.

- Antibiotic resistance due to over-prescribing, indiscriminate use in the poultry and farming industry has resulted in a veritable nightmare of untreatable infections coming to light. Trigger happy doctors and dentists tend to prescribe the antibiotics in a very lackadaisical pattern which is mostly counter productive. McGavock H Professor at University of Ulster has given a doomsday prophecy that most antibiotics effective today will become totally useless by the year 2015[11] and that patient may succumb to day-to-day infections which are easily treated today.
- Spurious drugs coming in Indian market has become a matter of increasing concern to the practicing dentist.

Rationale of Antibiotic Therapy

- Use one with narrowest spectrum that will target the suspected organism.
- Use the one with minimum toxicity as far as possible.
- Use the appropriate duration and strength.
- Provide concurrent analgesics.
- Combine with appropriate surgical intervention.
- Adequate patient monitoring and follow-up.

Antibiotic Abuse

Indiscriminate use of antibiotics for minor causes and improper dosing has resulted in emergence of resistant strains of microorganisms. This has become a major threat to cure of infectious diseases, e.g. tuberculosis (multidrug resistant T.B). Over the counter dispensing of antibiotics of higher strength for shorter period of time without doctors prescription is another important factor for development of antibiotic resistance in our country. In this scenario proper patient education about importance of duration and dosage is mandatory.

Commonly Used Antibiotics

Penicillin

Penicillin exerts a bactericidal action in susceptible bacteria by inhibiting cell wall synthesis.

Benzyl penicillin (Pentids ®) and benzathene penicillin (Pencom ®) have narrow spectrum of activity and is mainly active against gram + ve bacilli, both gram + ve and –ve cocci, and spirochetes.

Cloxacillin and Fluctoxacillin also have similar spectrum of activity but are also active against pencillinase producing organisms such as *Staph aureus*.

Amoxycillin (Pressmox®, Symoxyl®) and ampicillin (Rocillin ®) have broader spectrum of activity than benzyl penicillin and are active against much larger number of gram –ve organisms including *E.coli*.

Now-a-days a lot of combination of drugs are used to have wider rage of activity. Combination of cloxacillin with amoxycillin (Redclox®, Novoclox®) and ampicillin (ampiclox ®) provides wider range of activity. Clavulanic acid a beta-lactamase inhibitor when combined with a penicillinase-sensitive antibiotic (Augmentin ®) provides further wide range. Some combinations of amoxycillin have appeared in the market with Lactobacilli spores included. This reduces the chances of gastrointestinal tract disturbances due to decimation of the commensals.

Cephalosporine

They are the broad-spectrum bactericidal drugs that inhibit cell wall synthesis. They are effective against wide range of gram + ve and gram—ve organisms. The second and third generation cephalosporins are active against wide range of organisms including *H. influenzae, N. meningitides, N. gonorrhoae* and *P. mirabilis.*

Tetracycline

They are the broad-spectrum antibiotics very affective against most of the organisms that cause periodontal disease, e.g. gram + ve organisms like *Actinomyces*, peptostreptococci, lactobacilli and others like *B.gingivalis. A. actinomycetemcomitans.*

Tetracycline is shown to concentrate in gingival fluid and reported to have unique efficacy in juvenile periodontitis. Subantimicrobial doses of doxycycline have shown to be effective in long-term management of adult

periodontitis. Caton JG et al (2000)[1] have used 20 mg doxycycline t.i.d used for 9 months along with routine scaling and root planning in patients with adult periodontitis showed greater improvement in clinical attachment and probing depth (Fig. 36.1).

Minocycline

It is a semisynthetic broad-spectrum antimicrobial agent that was first introduced into clinical practice in 1967. The most common use of minocycline is for the long-term treatment of acne vulgaris. A well-recognized side effect of minocycline treatment is pigmentation, which has been reported in multiple tissues and fluids including thyroid, skin, nail beds, sclera, bone, and teeth. While there have been several reports of oral pigmentation following minocycline therapy, these have been, for the most part, pigmentation of the underlying bone with the overlying oral mucosa only appearing pigmented.[7] However Cascio A et al (2004)[8] reports no findings of dental defects in children treated with minocycline.

Locally administered time-released antibiotics targeting specific microorganisms may inhibit the action of collagenase, an extremely destructive enzyme that is released into periodontal tissues via the host response.[9] Paquette D et al (2003)[10] have proposed that treatment with scaling and root planing (SRP) plus locally delivered minocycline microspheres is more effective than SRP alone in reducing pocket depths in smokers with periodontitis (Cynomycin®).

Macrolides

They exert antibacterial effect by inhibiting bacterial protein synthesis. Erythromycin is used in patients with penicillin hypersensitivity.

Azithromycin 500 mg once a day is shown to effective in the management of acute periodontal abscess[2].

Roxithromycin 150-300 mg twice daily is effective in upper respiratory tract infections.

Amsden GW et al (1999)[6] recommend a dose of 1,500 mg per day in serious cellulites and where there is suspicion that gastrointestinal tract absorption is going to be reduced. 1,500 mg dose of oral azithromycin can be administered over either 5 or 3 days. Newer macrolides are extremely useful for dental practitioner but the cost remains prohibitively high.

ANALGESIC AND ANTI-INFLAMMATORY

Rational analgesic therapy requires an understanding of each patients specific needs and knowledge of the analgesics available for clinical use. Earlier analgesic therapy was based on the experience gained from treatment of previous patients articles in the literature, professional advertisements and other company promotional materials. Current literature provides the basis for scientific judgment on drugs, doses and combinations for optimal therapy.

Analgesics are broadly classified as opioids and non-opioids. The opioids usually act centrally and induce sleep. But the general tendency to group opioids as centrally acting and non-opioids as peripherally acting is eroded by the demonstration of peripheral effects of opioids in inflammatory conditions [3] and evidence for central effects of NSAIDs.

Opioids

Clinically opioids can be grouped into two categories depending on their efficacy.

i. Intravenously they have rapid onset.
ii. Orally administered opioids are less reliable in producing predictable sedation as considerable patients variation is noted. Low efficacy opioids are added to a NSAID to increase analgesic effect.

In dentistry the opioids are used mostly in oral surgery, road accidents and in cancer pain control. The comparison of some commonly used opioids is given below.

Generic name	*Dose*	*Duration of analgesia*	*Efficacy*	*Addiction*
Morphine	10mg	4-5 hrs	High	High
Methadone	10mg	4-6 hrs	High	High
Codeine	30-60mg	3-4 hrs	Low	Medium
Pentazocine	30-50mg	3-4 hrs	Medium	Low

Non-steroidal with Inflammatory Drugs (NSAIDs)

The use of non-steroidal anti-inflammatory drugs for acute dental pain is a genuine therapeutic improvement in that greater analgesic efficacy is seen than for single entity agents like aspirin and paracetamol. The NSAIDS block

prostaglandin synthesis by preventing the conversion of arachidonic acid, which is made available after cell injury, to the endoperoxidase. The analgesic and anti-inflammatory effects are thought to occur in the periphery while the anti-pyretic activity is a central nervous system effect mediated by the inhibition of pyrogen stimulated prostaglandin synthesis in the hypothalamus.

Commonly Used Drugs in Pain Control

Aspirin and Paracetamol

Aspirin is the most commonly used analgesic, anti-pyretic and anti-inflammatory agent. It is effective against the dull, throbbing mild to moderate type of pain of inflammation, in which locally synthesized arachidonic acid metabolites sensitize peripheral nerve endings to endogenously pain producing substances. The inhibitions of these metabolites occur at the site of tissue injury resulting in decrease in pain perception.

Paracetamol has analgesic and anti-pyretic effect that do not differ much from that of aspirin but for its weak anti-inflammatory action. The properties of the two can be compared as follows:

Property	*Paracetamol*	*Aspirin*
Analgesia	+	+
Anti-inflammatory	_	+
Anti-pyretic	+	+
Anti-thrombotic	_	+
Onset of action	30 min	30 min
Max daily dose		
Adult	3 gm	3.9 gm
Children	1.2 gm	Avoid in children

The best single dose of aspirin is that which sustains relief without causing toxicity. Paracetamol is a suitable substitute for aspirin for its analgesic or anti-pyretic effects in patients where aspirin is contraindicated. (e.g. prepartum patients, asthma, peptic ulcer, bleeding disorders and children with febrile conditions). However the general substitution of paracetamol for aspirin as an analgesic is not recommended.

Ibuprofen (Brufen ®): Ibuprofen is the prototype of the NSAID class of drugs and is useful in conditions where paracetamol or aspirin do not provide enough analgesia and use of opioid containing analgesics would likely to result in CNS or gastrointestinal side effects. Dose: 400-600 mg every four to six hourly not to exceed 3200 mg/day

Diclophenac: Diclophenac offers good analgesic, and anti-inflammatory action equivalent to ibuprofen. Available as diclophenac sodium (Voveran®) and diclophenac potassium (Voltaflam®) for oral, parenteral and topical use. Dose: 50mg bid orally.

Piroxicam (Dolonex®): It is an oxicam NSAID, its plasma half-life is estimated at 4-5 hours which allows once day dosing. It is suitable for use as short-term analgesic and long-term anti-inflammatory drug. Dose: 20 mg BD initially followed by 20 mg OD.

Ketarolac (Ketanov®):It is the first NSAID approved for intramuscular administration for the short-term management of moderate to severe pain and efficacy is equal to that of morphine. Dose: 15-30 mg i.m., every 4-6 hourly (max 120 mg/day). Orally it is used at a dose of 10-20 mg 6 hourly for short-term management for moderate pain. Continuous use for more than 5 day is presently not recommended.

COX-II Inhibitors

It is generally believed that two forms of cyclo-oxygenase enzymes are responsible for the arachidonic acid cascade. One form COX-I is responsible for the normal homeostatic of prostaglandins in the GIT. The form COX-II is believed to be induced only during inflammation and contribute to pain and edema. The NSAIDs usually act by suppressing activity of both COX-I and COX-II isoforms of cyclo-oxygenase. Observations that the COX-1 is distributed throughout the body, whereas COX –2 expressions are limited to a few specialized tissues and is induced during inflammation, lead to the hypothesis that COX-1 is primarily responsible for the adverse GIT effects of existing COX-1/ COX-2 inhibitors. This hypothesis suggests that the regular NSAIDS with dual COX-I/COX-2 inhibitors produce both therapeutic and toxic effects, whereas selective COX-2 inhibitors should have therapeutic effects largely devoid of NSAID toxicity.

Nimesulide (Nalgis®, Remulide®, Nimbid®): It is a relatively week inhibitor of PG synthesis used primarily for short lasting painful inflammatory conditions like sport

injury, sinusitis, postoperative pain and arthritis. Most asthmatics and those who develop bronchospasm or intolerance to aspirin and other NSAID do not cross react to nimesulide. Its specific usefulness appears to be only in such patients.Dose: 100 mg BD.

Rofecoxib (Rofica®, Rofact®) and Celecoxib are two drugs today at market which has high selectivity for COX-2 inhibition. Single doses of celecoxib were demonstrated in oral surgery model of acute pain and it was comparable to that of 650 mg aspirin. Similar study model have demonstrated rofecoxib as having better analgesic effect than celecoxib. The 25 mg and 50mg doses of Rofecoxib were shown numerically superior but statistically indistinguishable from naproxen.[4.]

***Rofica Plus®*:** A combination of Roficoxib with paracetamol is also found to be effective in controlling mild to moderate odontogeniic pain.

Valdecoxib (Bextra tablets of 10 mg and 20 mg) is a new COX-2 inhibitor.It is a isoform of cyclo-oxygenase. It is indicated for the symptomatic treatment of osteoarthritis or rheumatoid arthritis (10 to 20 mg once a day) and for the treatment of primary dysmenorrhea (40 mg once a day). Valdecoxib is as efficacious as conventional non-COX-2 selective NSAIDs, but offers the advantage of a much better gastrointestinal tolerance. Valdecoxib has a prodrug that can be administered intravenously or intramuscularly (Parecoxib, Dynastat) and has been developed for the short-term treatment of postsurgical pain.[12]

Etoricoxib (Etrobax® 60/ 90/ 120mg): It is a new COX-2 inhibitor with reduced GIT side effects. Dosage of 120 mg OD is indicated for acute pain control. It is better avoided in children and in pregnant women.

Though the news elective COX-2 inhibitors like rofecoxib holds promise for the analgesic efficacy with greater safety than ibuprofen and related NSAIDs, it is prudent to wait until additional clinical experience for safety is obtained.

ANXIOLYTICS

Anxiety that occur in patients without any apparent exogenous cause need help. Non-drug therapy may be the best for many but drugs are often useful for patients having high level of anxiety, and for tiding over that crisis.

Benzodiazepines: Discovered in 1960, they are the drugs of choice for treating anxiety conditions. Their primary advantage is the suppression of anxiety without producing overt sedation or even unconsciousness, typical of other CNS depressants. It should be noted benzodiazepines should not be used as antidepressants, except perhaps the Alaprazolam when anxiety is dominant in mixed anxiety/depression conditions.

Anti-depressants: These are the drugs that elevate mood in depressive illness.

Tricyclic anti-depressants are used in dentistry in patients with depressed but not agitated psychotics. They can be grouped as:

1. *Noradrenaline and seronin reuptake inhibitors*: Imipramine, amitriptyline, trimipramine,clomipramine
2. *Noradrenaline reuptake inhibitors:* Amoxapine, nortriptyline, desipramine,
3. *Selective serotonin reuptake inhibitors*: Fluoxetene, fluvoxamine, paroxetine
4. *Atypical anti-depressants*: Tradozone, brupopion, tianeptine

Clinical Situations

- A twenty-two-year old healthy female comes with a grossly carious mandibular molar with radiographically seen periapical abscess. She refuses any endodontic care. Since the patient is immunocompetent and the since the tooth extraction under aspectic condition provides sufficient drainage, the prescription can be limited to an analgesic like Ibuprofen 400 mg three times a day for period of three days.
- A forty-five-year old male who is known diabetic since last three years comes with grossly caried wisdom tooth and pericoronitis. Intraoral periapical radiograph reveals mesioangular impaction, periapical abscess, and clinically pericoronal suppuration is also evident. Serum glucose level taken in the morning was 133 mg percent. The option that we recommend is 1 gm of amoxycillin two hours prior to removal of the wisdom

tooth by either open or regular method. Suitable analgesic preferably a combination like Imol® (Paracetamol + ibuprofen) offers a slight advantage since the extensive infection might need anti-inflammatory and anti-pyretic care. The antibiotic in this case is best given for upto 5 days total at least 1500 mg/day and analgesics are given t.i.d for the first day and 1 SOS after that.

- A fifty-five-year old male comes with multiple periodontal abscesses. His serum glucose level is found to be within normal limits. After performing a routine oral prophylaxis the prescription can have doxycycline 100 mg per day with a starter dose of 2 such capsules; for a period of seven days. Further referral to the periodontist is recommended.
- A twenty-three-year old female patient comes with chief complaint of preauricular pain in both the sides of the face. Pain is radiated to the masseter and temporalis region bilaterally. The soreness of all the facial muscles is more early in the morning. The facets of molar and premolar teeth are ground flat. The dentist records clicking sounds on both TMJs and makes a tentative diagnosis of MPDS. She has been married two years back and her husband has been working in Dubai for last two years. This problem also originated since last one and half years. Establishing the psychosomatic basis of this problem, the dentist makes a occlusal splint and prescribes Brufen® 400 mg tid for one week for acute pain and Depsonil-DZ®(Imipramine 25 mg and Diazepam 2 mg). Three times a day for one month. Patient is recalled and regularly counseled and asked to take up hobbies to use the time constructively.
- A fifty-eight-year old female with CC of severe burning sensation on the oral cavity since two years is extensively investigated for CBC and random serum glucose and both are determined to be normal. No changes are visible on tongue and mucosa. Personal history reveals that her husband expired two years back and her only son is settled in USA. A tentative diagnosis of BMS or burning mouth syndrome is made and dentist prescribes her Tab Anxit® (Alprazolam 0.25 mg) three times a day for one month before further evaluation. She is also referred to a friendly clinical psychologist for systematic counseling.

FIGURE 36.1: Showing a 28-year-old male who was on prescription Nefidepine since last one year. He developed a tough fibrous type of gingival enlargement since last three months. Flap surgery with doxycycline prescription resulted in better gingival health and esthetics (Courtesy Bailoor DN, Rohit Malik, Sunil, Aruna N, 2001 Yenepoya Dental College Hospital,Mangalore)

CONCLUSION

It is important to be vigilant regarding side effects and abuse of any drug before using them. With the availability of newer and better drugs, the clinician is definitely at better advantage while combating diseases. With newer brands available, the commercial marketing strategies of the drug companies may attract the clinician. But the treatment of diseases with drugs in every single patient should be carefully evaluated. The issue of side effects regarding medication used and the risk versus benefit for each case should be considered. It is definitely wiser to take expert opinion from an oral medicine specialist when in doubt regarding the use of a drug.

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This chapter is not a replacement for professional dental training. Kindly verify the latest prescribing practices with your teachers and consultants prior to making real life decisions. Most values are indicative and have been checked against latest reliable sources, but the publishers and editors do not have any direct or indirect liability to the use or misuse of this prescribing information.

Prior to prescribing any medication please check that they are from ethical drug manufacturers following sound quality control practices. Follow the manufacturers directions in most prescriptions and in case of new drugs please confirm side effects, safety in children and pregnancy with the nearby-approved University hospital specialists and legitimate inter-net sources.

—Editors

B Sureshchandra, Vijay Singh S,
Verma Ravi, Pradeep CV

37 Pulpal Pathosis

INTRODUCTION

The dental pulp in its unique environment has been a tissue of mystery since ages. It behaves strangely and responds unpredictably to various stimuli and treatment. This variability of pulp response has led clinicians to contradictory statements that "the pulp if injured is a dying pulp", while others with quite an opposite remark that "the pulp cannot be killed with an axe". Neither statement is correct and the answer lies somewhere in the midway of the two.

When the pulp is injured, it responds with inflammation. This in turn increases the vascular permeability and fluid leakage into the surrounding tissue. As the pulp is entirely enclosed within an unyielding chamber of dentin; hence it has no space to swell unlike other soft tissues and this leads to cell death. Moreover, lack of collateral blood supply of the coronal pulp makes it highly compromised in its ability to defend itself from severe irritation.

Depending on the severity and duration of the insult and the capacity of the host to respond, the pulpal response ranges from transient inflammation to irreversible pulpitis to total necrosis of the pulp. It is up to the clinician to decide whether the pulp has been reversibly or irreversibly damaged and to manage it accordingly.

PAIN PERCEPTION IN THE PULP

The neurons, which innervate the dental pulp, have cell bodies in the trigeminal ganglion, and the central synaptic connection extends upto the subnucleus caudalis part of trigeminal spinal track nucleus, which is specifically involved in pain and temperature transmission.

The Pulpal Pain Fibers (nociceptive) consists of two types—A delta (d) fibers and C-fibers (Fig. 37.1).

A Delta () Fibers

They are myelinated and have a diameter of 2 to 5 µm, with electric impulse travelling along the nerve at a speed

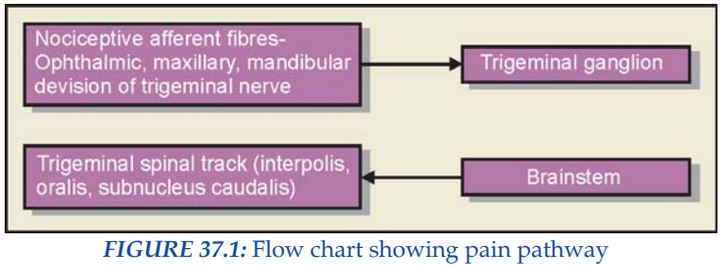

FIGURE 37.1: Flow chart showing pain pathway

of 5 to 30 m/sec. The terminals of the nerve are located superficially in the dentinoblastic and subdentinoblastic zones. They have a relatively low threshold to stimulation and can be stimulated even without damaging the tissues. The pain response is immediate, sharp and localized.

C-fibers

They are unmyelinated and have a diameter of 0.4 to 1.0 µm, with the electric impulse traveling along the nerve at a speed of 0.4 to 2 m/sec. The terminals of nerve are located near the blood vessels throughout the pulp. They require a higher threshold for stimulation and intensity of stimulus should be capable for tissue damage. The pain response is delayed, dull and radiating. Culbreath TE et al[8] 2000.

The late appearance of the A fibers in young pulp explains why the entire pulp test tends to be unreliable in young teeth. The peripheral mechanisms of pain are much more complex, and differ from acutely injured normal tissues compared with chronic inflammation or neuropathic pain Byers M R and Narhi MV (1999).[6]

DIAGNOSIS OF PULP PATHOSIS

The recognition and naming of pulp disease is based on accurate history, proper clinical examination and selective investigations.

The selective investigations include:

- Intra oral periapical radiograph.
- Pulp vitality testing.
- Fiber optic transillumination.
- Pulp flow cytometry.
- Digital imaging.
- Pulse oximetry or laser Doppler analysis.
- Ortho-CT

CLASSIFICATION OF PULP PATHOSIS

I. Inflammatory diseases of Dental Pulp
II. Reversible Pulpitis. (Hyperalgesia)
   - Hypersensitive dentin
   - Hyperemia.
III. Irreversible pulpitis
   - *Painful Pulpitis*:
   - Acute Pulpitis. (Acute Pulpalgia)
   - Subacute Pulpitis (Chronic Pulpalgia)
   - *Non-Painful Pulpitis.*
   - Pulp Polyp (Chronic Hyperplastic pulpitis)
   - Chronic Ulcerative pulpitis.(Open form)
   - Chronic Pulpitis (closed form)
IV. Other Pulpal Changes
   - Necrosis
   - Degenerative (Retrogressive) Changes
   - Atrophic pulposis.
   - Calcific Pulposis (Dystrophic Calcification)
V. Internal resorption

INFLAMMATORY DISEASES OF PULP

Reversible Pulpitis

It is a pulpal inflammatory condition in which the pulp is capable of returning to its healthy state after the removal of noxious stimuli. Pain is of short duration and may be produced by external stimuli, such as touch, cold, heat, air blast, etc. Patient may also give a history of recent restorative procedure. Pain usually subsides as soon as the stimulus is removed. The pain is caused by the stimulation of A fibers (Lower threshold fibers). Culbreath TE et al (2000).[8]

Histologically it may represent a response ranging from hyperemia to mild to moderate inflammatory changes.

Hypersensitive Dentin

Brannstrom Met al (1967)[4] introduced Hydrodynamic theory, in which dentin pain and odontoblastic displacement were related. During cavity preparation, pressure or thermal change causes displacement of content and odontoblastic tubules resulting in pain. Brannstrom M (1981).[5]

Dessication of dentin by blast off air can cause dentinal fluid to flow outward at the rate of 2 to 3 mm/sec. Linden LA, and Brannstrom M (1967).[12] Moreover hyperosmotic concentration of sucrose or calcium chloride when applied on exposed dentinal surface produces pain Lilja et al (1982).[11] Acid etching of dentin is also capable of increased fluid flow resulting in increased dentin sensitivity. The dentinal fluid expands or contracts when stimulated. Since the coefficient of expansion of the fluid is greater than

dentin, there is movement of dentinal fluid either internally or externally in response to stimuli. Culbreath TE et al[8] 2000.

It is the A fibers rather than C fibers that are activated by stimuli such as heat, cold, and air blast when applied to exposed dentin. Prolonged vasodilation or local incipient inflammation may result in lower pain threshold of peripheral receptors which may be another factor for hypersensitive dentin Weine FS (1996).[24]

Diagnosis: The patient gives the history of pain, which is sharp, of short duration and subsides when the external stimuli is removed. Periodontal space and lamina dura are normal radiographically. The tooth is normal on percussion and the patient responds more to cold stimuli. Electric pulp testing may elict positive response with minimum current.

Treatment: Hypersensitive dentin can also be treated by reducing the diameter of dentinal tubules, so that fluid movement is restricted. This can be achieved by using dentin bonding agents to seal the tubules, use of oxalate compounds to form insoluble ppts, which occludes the tubules, dentinal tubules impregnated with resins or formation of smear layer on the sensitive dentin by burnishing the exposed root surface. Trowbridge HO (1990).[22]

A significant reduction in sensitivity and pain was observed by burnishing saturated potassium nitrate solution. Touyz LZ and SternJ (1999).[20]

Hyperemia

Hyperemia is an initial pulpal response, which is reversible and is the beginning of the inflammatory cycle. There is engorgement of capillary bed leading to edema. The intrapulpal pressure (normally 8-15 mm Hg) increases because of increased blood volume in rigid encased environment of the dental pulp.[24]

Enamel and Cementum normally act as impermeable barrier, but breakage of the barrier by caries or restorative procedure makes the dentinal tubules bidirectional. This leads in outward flow of dentinal fluid due to increased intrapulpal pressure and there is diffusion of external toxic substances into the pulp.

The presence of hypersensitive dentin does not mean that hyperemia is present, although hyperemia if present may be accompanied with hypersensitive dentin (Fig. 37.2).

Diagnosis: The patient gives a history of Pain when in contact to external stimuli. Pain is sharp, of short duration and subsides after the stimulus is removed. Visual examination may show, caries, restoration, traumatic occlusion, or fracture. Radiographic picture shows the periodontal ligament space and lamina dura to be normal.

On percussion the tooth is normal. The tooth responds more than normal to cold stimuli, and electric pulp testing may elict positive response with minimum current.

Treatment: Prevention is the best treatment of reversible pulpitis. Routine examination for caries, proper cavity

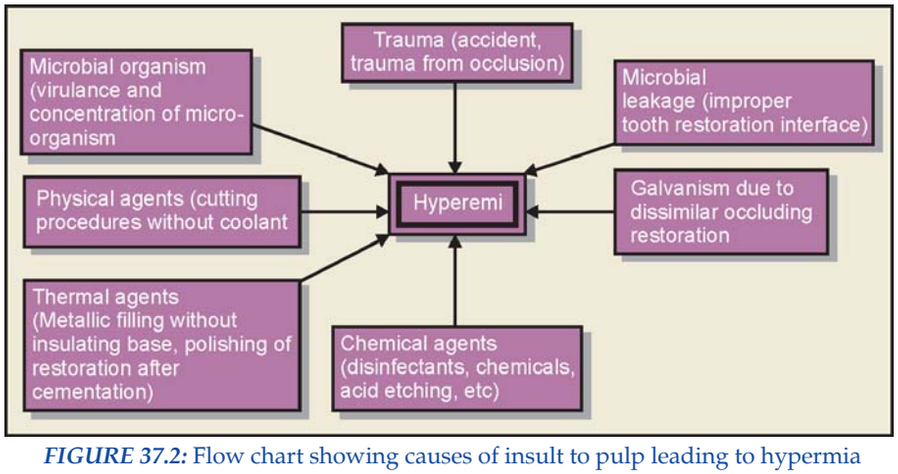

FIGURE 37.2: Flow chart showing causes of insult to pulp leading to hypermia

preparation with use of coolant and use of base in deep cavity to prevent thermal insult to the pulp is to be carried so that pulp does not become hyperemic. Chemical irritants and acid cements are also avoided in deep cavities.

Irreversible Pulpitis

Painful Pulpitis

It is a clinically detectable response of pulp to irritant. There is an increase in exudation factors, which increases the intrapulpal pressure beyond the threshold limit of pain fibers resulting in Pain.

Madisons (1989)[14] has been shown that leukoterine B4 to potentiates nociceptor activity during pulpal inflammation. Olgart L (1986)[17] has stated that moreover bradykinin and histamine can produce dull aching pain in deep cavities of human teeth. Ohnishi T et al (2000)[16] have commented that Hepatocyte growth factor (HGF) which is a broad spectrum and multifunctional cytokine, has been shown to be involved in the development and regeneration of various tissues including dental pulp and also increases during acute inflammation of tissues. Barkhordar RA et al (1999)[2] have indicated that cytokine interleukin-6, a major mediator of host response to tissue injury and infection, which has been detected, in human dental pulp and periapical lesions (Fig. 37.3).

Acute Pulpitis

The acute inflammatory response in acute pulpitis is irreversible and characterized by severe pain even after removal of stimuli. The inflammatory response leads to vasodilation, inflammatory oedema, leukocytic infiltration followed by pulpal abscess.

Subacute Pulpitis

Subacute pulpitis also known as chronic pulpalgia is a mild exacerbation of chronic pulpitis. It is characterized by mild to moderate pain induced by transient pressure from exudation.

The etiological factors are similar to hyperemia, but may also develop as an acute exacerbation of previously, non-painful, chronically inflamed pulp, for example, when food is impacted in a carious exposed pulp resulting in blockage of drainage or operative procedure resulting in stimulation of acute response in a chronic inflamed non painful pulpitis.

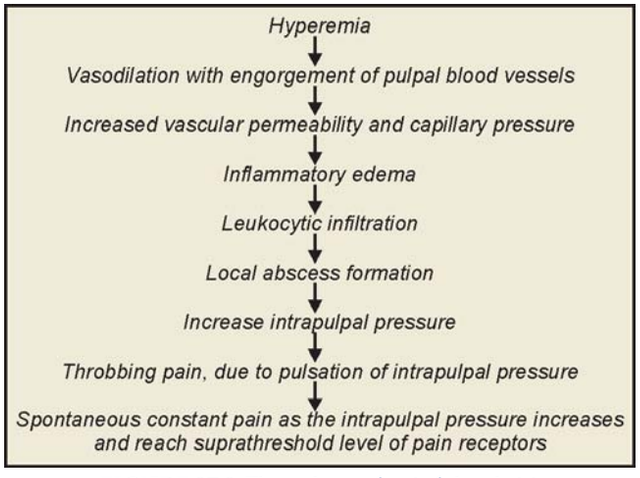

FIGURE 37.3: Flow chart of painful pulpitis

Even a slight increase in pulpal fluid volume will raise the tissue pressure, which may compress the blood vessels, leading to ischemia and necrosis. However, the increased tissue pressure may in turn increase lymph flow and absorption of fluid into capillaries in nearby non-inflamed tissues. Both of these factors will transport fluid out of the affected area and subsequently out of the tooth and thus, lowering the tissue pressure. Heyeraal K J and Berggreen E [10]1999 states that the increased tissue pressure will cause outward flow of fluid through exposed dentinal tubules and help to protect pulp against entry of harmful substances.

Diagnosis of Painful Pulpitis

In case of acute pulpitis pain persists even after removal of primary irritant. The pain is usually diffused and not readily localized by the patient. On lying down the pain increases as the intra-pulpal pressure increases.

In sub acute pulpitis the pain is diffused, moderate referred and difficult to localize. The episodes of pain are intermittent.

Visual examination may reveal caries or restoration. Radiographically the periapex is normal, however, slight widening may be seen in advanced stage of painful pulpitis. Tenderness on percussion may be present due to increase in intrapulpal pressure as a result of inflammatory exudates.

Heat will increase pain as blood vessels, tissue and gaseous products of proteolysis expand, whereas cold will reduce pain in advance stage of pulpitis because of contraction of vascular bed, reducing the intrapulpal pressure.

Culbreath TE et al (2000)[8] states that the electric pulp testing responds to low current only in early stage when A fibers are viable, but in later stage more current is required as A fibers are destroyed.

Non-painful Pulpitis

It is an inflammatory pulpal response of pulpal connective tissue to irritant. There is a decrease in the exudative inflammatory activity resulting in decrease in intrapulpal pressure below the threshold limit of pain receptor (Fig. 37.4).

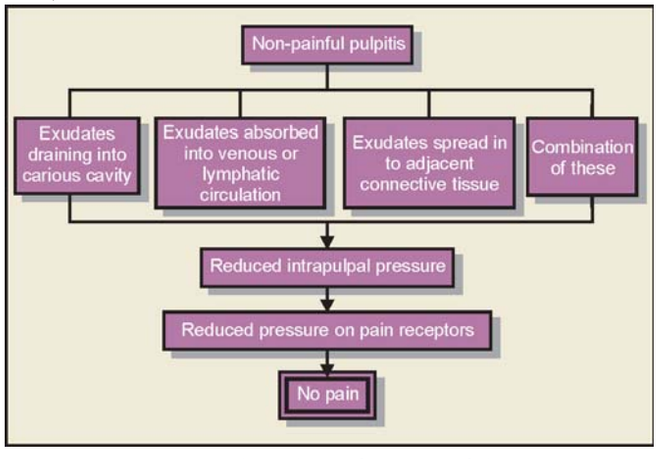

FIGURE 37.4: Flow chart of non-painful pulpitis

PULP POLYP (Chronic hyperplastic pulpitis)

It is a chronic inflammation due to a large extensive carious exposure of young pulp in an attempt to heal and repair. It is characterized by the development of granulation tissue lined by stratified squamous epithelium and results from low grade, long standing irritation. According to Grossman L[9] 1981 The epithelium may be derived from the gingiva or from freshly desquamated epithelial cells of the mucosa or tongue. The granulation tissue is vascular, contains polymorphonuclear neutrophils, lymphocytes and plasma cells. It is not painful to touch because of decreased number of nerve fibers, however, because of the vascularity it bleeds easily. It is seen in children, adolescents and is freshly reddish pulpal mass that fills the cavity or pulp chamber.

Treatment of pulp polyp is to curette with sharp instrument and later on followed by pulpectomy.

Chronic pulpitis (closed form)

The closed form of chronic pulpitis may occur due to trauma, restorative procedure, orthodontic tooth movement or periodontal lesion, but carious lesion is absent.

The pulpitis may be chronic from onset or become chronic after the acute response subsides depending upon the strength and duration of irritant. Additional operative procedures may cause exacerbation of the condition. According to Weine F.S.[24] 1996 Resolution of chronic pulpitis may decrease and obstruct the lumen of pulp chamber and canal because of formation of denticles, diffused calcification and irritation dentin (Fig. 37.13).

Chronic pulpitis (ulcerative or open form)

In open form of chronic pulpitis the pulp is cariously exposed and is characterized by the formation of abscess at the point of exposure. Depending upon the involvement the chronic inflammation may be partial or total (Fig. 37.12).

Following zones are present:

Zone I: Zone of necrosis or infection.

Zone II: Zone of contamination. This zone surrounds the zone I and contains inflammatory components.

Zone III: Zone of irritation. There is dilution of irritant and repair and healing of tissues takes place. Young fibroblasts and new capillaries develop with formation of granulation tissue.

In chronic pulpitis, as there is adequate drainage, the intrapulpal pressure does not rise, therefore, pain is absent, however pain may develop if there is interference of the drainage by impaction of food. Pain also may develop if granulation tissue is contaminated.

OTHER PULPAL CHANGES

Necrosis

Stanley H R (1984)[19] states that Necrosis or death of pulp tissue may be partial or total and is a sequelae to inflammatory reaction of the pulp, but also occurs following a traumatic injury after which there follows an

FIGURES 37.5A and B: A 24-year-old female presented with severe pain in the maxillary anterior region with long standing caries, both the incisors were nonvital. There was tenderness on vertical percussion and radiographically irregular bone loss. diagnosis periapical abscess in relation to both the anteriors—endodontic therapy was initiated (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

immediate stoppage of blood supply leading to gangrenous necrotic pulp. Two types of Necrosis are observed:

1. Liquefaction necrosis.
2. Coagulation necrosis.

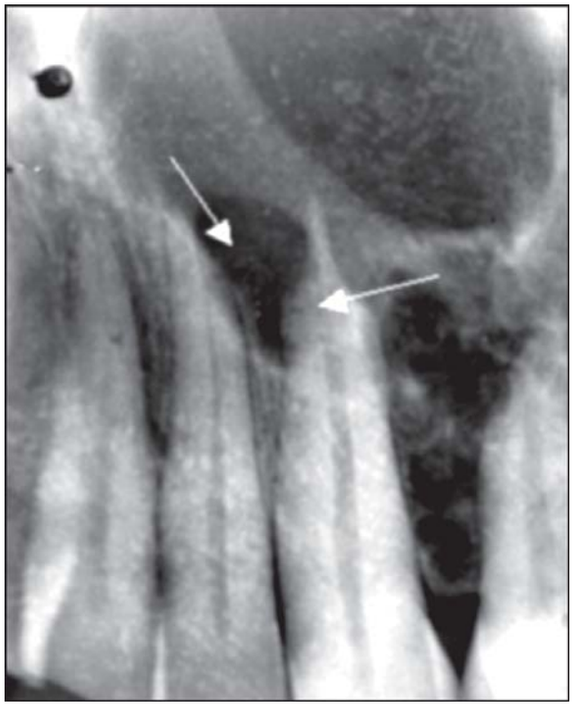

FIGURE 37.6: External Resorption from both the sides of the root apex giving an appearance of spine on the tip of the root. (Beena K, Omal PM, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

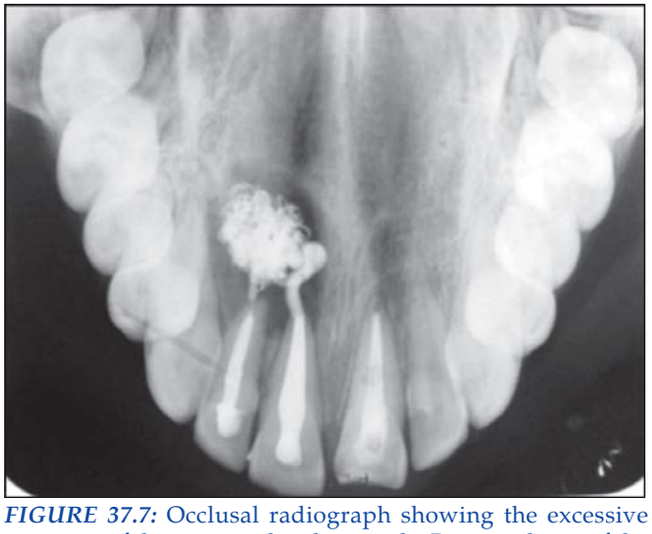

FIGURE 37.7: Occlusal radiograph showing the excessive extrusion of the root canal sealer into the Periapical area of the 11, 12. Such iatrogenic lesions may also precipitate giant cell reaction in the bone (Sunil PM, Aruna N, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

In liquefaction necrosis the proteolytic enzymes have softened and liquefied the tissues resulting in the formation

of pus. There is a good blood supply in case of liquefaction necrosis, but in case of coagulation necrosis, there is poor blood supply and the tissue has the appearance of cheesy mass composed of coagulated proteins, fats and water (Figs 37.5 and 37.7).

The end product of necrosis (Hydrogen sulphide, Ammonia, Fatty acids, Exotoxins, Endotoxins, Indole, Ptomaines, etc.) are toxic and may lead to inflammatory response of periapical tissue (Figs 37.6 and 37.9).

Diagnosis

Tooth discoloration may be seen because of hemolysis of RBCs, pain, swelling and response to percussion is not present unless the periapical tissue is involved. The radiographic finding is normal if there is no periapical involvement.

Vitality test is usually negative, except in liquefaction necrosis which may sometimes give positive electric response, moreover multirooted tooth may show mixed response as only one canal may show total necrosis.

Endo-antral Syndrome

Selden HS (1999)[25] has mentioned caution in endodontic treatment of maxillary posterior teeth. The spread of dental infection into adjacent maxillary sinus from periapical infection and abscess of maxillary premolar and molar teeth is termed as the endo-antral syndrome. The paranasal sinuses radiograph and in recent times the CT Scan helps in establishing unequivocally the fact that infection has spread from the periapical region.

A Caldwell—luc approach to the antrum is required in advanced cases of suppuration.

Degenerative (Retrogressive) Changes

Atrophic Pulposis

As the tooth grows older, atrophic (decrease in size of an organ) changes occur slowly. The size and the number of pulp cells decreases with increase in mature collagen fibers, this increase in collagen fibers is known as fibrosis. Seltzer et al (1963)[18] points out that atrophy of the pulp normally occurs with advancing age.

In case of severe attrition or abrasion, the hyper activity of stimulation because of irritation may increase the aging process (induced aging), this may decrease the size of canal by formation of irritation dentin, moreover the apical canal and foramina may reduce in size because of deposition of cementum in the periapex to compensate the occlusal wear, thus leading to altered blood supply to the pulp. The decreased vascularity makes the pulp more dehydrated and viscous. Moreover as more and more mature collagen is formed, it accumulates to give a crabgrass appearance. The defence mechanism is thus, reduced due to decreased vascular supply of proper oxygen and nutrition of the pulp. Thus the chronological age of the patient is not an indication of atrophic change to pulp as a young patient with severe irritation may have atrophic pulpal changes as well.

Calcifications

The incidence of calcification of pulp is common and is found in both healthy and aging pulp, and the incidence increases with age. Moreover the calcification also increases in size and density as the irritation increases. Linden LA and Brannstrom M (1967)[12] mentions that the calcifications are usually seen at the site of necrosis or along the blood vessels. The calcifications appear as an irregular calcific deposit and are known as diffused calcifications. Diffused calcifications are usually found in the root canals and less often in the coronal area (Fig. 37.8).

FIGURE 37.8: IOPA depicting a calcification in the pulp chamber of first molar and un natural dipping of the maxillary antrum interdentally. (Varma R, Pradeep CV, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

Deposition of calcium salts in dead or degenerating tissue results in dystrophic calcification, which may be due to alkalinity of destroyed tissue that attracts the salts. They may be seen in minute areas of pulp tissue affected by minor blood disturbances or around single degenerating cell.

The pulp stones or denticles are larger, well outlined calcification and seen more commonly in pulp chamber.

On the basis of location they may be classified as free, embedded and attached, and on the basis of structure may be true or false.

The pulp stones or denticles can be true denticles or false denticles. In case of true denticles the structure is similar to dentin and are usually located in the apical foramen. It is thought that true denticles are caused by inclusion of remnants of the epithelial root sheath within the pulp, whereas false denticles appear as concentric layer of calcified tissue and does not resemble the dentin. In the center of false denticles necrotic and calcified remnants may be present. Bhaskar SN (1990)[3] mentions that the nidi for calcification in false denticles may arise from calcification of thrombii in blood vessels, called Phleboliths. Van Den Berghe JM et al (1999).[23] Cases have been reported of presence of pulp stones throughout the dentition of monozygotic twins, and that, the pattern of calcification was partially consistent with the hereditary condition of dentinal dysplasia.

Pulp stones lying in the canal orifice or root canal may cause difficulty to locate the canal during endodontic thera.

FIGURES 37.9A and B: Showing the iopa radiographs depicting the external resorption (Nillofer S, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore)

INTERNAL RESORPTION (INTERNAL GRANULOMA, ODONTOCLASTOMA, PINK TOOTH)

According to Andreasen JO (1981).[1] Internal resorption is an idiopathic slow or fast progressive resorptive process occurring in the dentin of pulp chamber or root canal of teeth, and is characterized by oval shape, enlargement of root canal space. Ne RF et al (1999)[15] mentions two types of internal resorption—root canal (internal) replacement resorption and internal inflammatory resorption. Tronstad L [21]1984 mentions that the etiology is unknown, but it is thought that it is the pulp tissue that is inflamed as a result of infected coronal pulp space and communication between necrotic tissue and vital pulp through dentinal tubules, moreover in procedure like pulpotomies, strong chemicals that are used can predispose the canal to internal resorption according to Cohen S, Burns R (1994)[7].

It is asymptomatic and pain develops when the resorptive process extends to the periodontal ligament. It is usually detected during routine radiographic examination. The involved tooth may present as a pinkish hue in the crown because of resorption of coronal portion thinning the enamel and presence of capillary granulation. Microscopic examination shows scalloped dentinal wall with multinucleated dentinoclasts. Root canal treatment should follow when internal resorption is detected (Figs 37.10 and 37.11).

FIGURE 37.10: Occlusal radiograph depicting the internal resorption, cause not known. No history of trauma, infection or any pathology was given by the patient. In some cases trauma from occlusion may precipitate these changes (Beena K, Omal PM, Bailoor DN 2003 Yenepoya Dental College and Hospital, Mangalore)

Recently dual cure syringable composite resin in conjunction with a bonding agent has been used in treatment of internal resorption by Culbreath TE et al (2000).[8] This technique seals the dentinal tubules and strengthens the remaining tooth structure, also improving the outcome of resorptive defects and reduces operator's chair time.

FIGURE 37.11: *A* 17-year-old male came with complaint of severe pain in a diminutive size premolar. History of cricket ball injury was elicited. Radiograph revealed large pulp chamber and resorbed root (Prasanna K, Omal PM, Bailoor DN 2004 Yenepoya Dental College and Hospital, Mangalore)

Nguyen HQ et al (1996)[29] mentioned about utility of Electronic apex locators which are frequently used attached to a small size endodontic file. They concluded that the Root ZX indicated the location of an apical constriction even when the anatomic constriction was eliminated. In the enlarged canals, length measurements obtained with small and large size files were comparable.

Abrahams and Hayt (1999)[28] developed dental CT with multiplanar reconstruction of the jaws (DentaScan)® which is useful in showing any relationship between apices of teeth and the adjacent anatomical structures.

FIGURE 37.12: An 11-year-old male patient presented with fractured left central incisor 21 due to cricket ball injury about 2 years back. 11 was also discolored but painless. Radiographically a clear radiolucent area measuring 2 cm × 1 cm was seen and the circumscribed radiopaque linear shadow of the cyst was evident. 11 also showed a Blunderbus apex and the roots were flaring due to the pressure of the lesion (Beena K, Omal PM, Bailoor DN 2003 Yenepoya Dental College and Hospital, Mangalore)

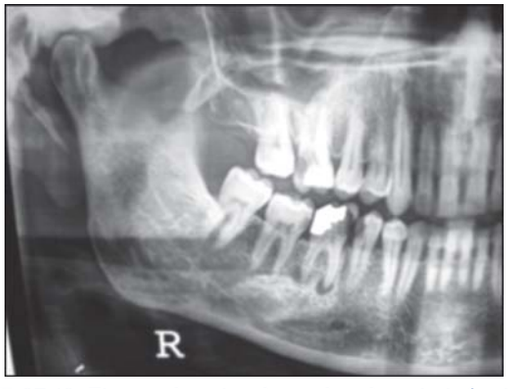

FIGURE 37.13: Figure showing how the chronic infection of the pulp and the periapical lesion can cause radiopaque osteosclerotic reaction around the periapical bone. This walling off effect is usually body's reaction to low grade chronic infection. Umarji H, John Ani, GDC Mumbai 2003

Another similar technique developed in Germany by Solar and Gahleitner (1999)[27] have developed Dental CT which is Dental computer assisted tomography. This represents a valuable addition to the diagnostic spectrum for oral and maxillo-facial regions. The possibility to transmit pictures electronically or on hardcopy without loss of quality is another positive point.

Recent imaging technique termed as ortho-CT was developed in Japan by Terakado M et al[26] 2000. Ortho cubic super-high resolution computed tomography (Ortho-CT), which they developed had following advantages:

i. Small size of the unit
ii. Ability to produce 3-dimensional images of high resolution
iii. Low-radiation doses. This can take high-resolution 3-dimensional images at any tomographic layer with only 1 exposure. They found this machine extremely useful for delineating periapical lesions, fractures in the roots and imaging of supernumeraries.

SUMMARY

The dental pulp is a complex and unique tissue. The diagnosis of pulpal disease needs a thorough understanding of the anatomy, physiology and the pulpal pathogenesis. As the signs and symptoms often tend to overlap, the differential diagnosis is quite an important aid to the operator in successfully treating the patients.

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BH Sripathi Rao, Bailoor DN

38 Infections of the Oral Cavity

INTRODUCTION

The Oral Flora: The oral cavity is a dynamic microbiologic eco-system. From childhood to old age the numbers and types of organism that populate the mouth vary continuously. There are unique ecologic niches like tongue (*Streptococcus salivarius*), tooth surfaces (*Streptococcus mitis*, *Actinomyces viscosus*) and in anaerobic gingival crevice (*Bacteroides*, *Spirochetes*) are colonized. Even within an age group factors like diet, smoking, antibiotic therapy, pregnancy and racial factors may influence composition of bacterial population (Fig. 38.1).

The groups of bacteria like *Streptococcus*, *Veillonella*, *Lactobacillus*, *Corynebacterium*, and *Actinomyces* accounts for more than half the oral flora. These exist naturally and are termed as Commensals. When the immunity of the person goes down, these commensals can attack and become pathogenic.

On the hard surface of the teeth proteins from saliva are deposited and organic film called pellicle is formed. Bacteria colonize this pellicle and it can form into a loose aggregation called materia alba or a well-organized colony termed as dental plaque. Materia alba is a whitish curd like aggregation, which can be removed easily by a water spray. It normally contains groups of bacteria leukocytes and desquamated epithelial cells. There is no organized structure for this.

Dental plaque is a complex bacterial colony, which helps members to survive by metabolic interdependence. We classify plaque into subgingival and supragingival on basis of its relationship to the margin of gingiva. These are likely to be the starting points for the initiation of periodontal disease (Fig. 38.2).

S. mutans is one of the primary etiologic agents for dental caries.

i. It ferments sugars to lactic acid.
ii. It produces extra cellular dextrans which sticks the bacteria to tooth surface.

Pit and fissure and smooth surface caries are attributed to *S. mutans* and *Lactobacillus* species. The dentinal caries are attributed to *Lactobacillus* species and *Actinomyces naeslundii*. Filamentous rods, *Actinomyces viscosus* and *Actinomyces naeslundii* cause the root caries.

The treatment of dental caries and periodontal diseases are specialities in themselves. We shall describe the management of some common infections.

Whenever any organisms interact with the host, two important factors come into play, immunocompetency of the individual and the virulence of the organism. The infections themselves could be specific like syphilis, gonorrhea, and Tuberculosis; or they could be non-specific like Acute necrotizing ulcerative gingivo-stomatis in which a random mixture of fuso-spirochaetal organism may

attack the host. Osteomylitis is another example of a mixed infection. Most odontogenic infections are mixed in nature and predominantly anerobic (35% aerobes and 65% anaerobes) Mixture may contain any of the following species—*Peptostreptococcus*, *Bacteroides*, *Fusobacterium*, *Streptococcus milleri*, Diphtheroids, *Actinomyces*, and many more commensal organisms.

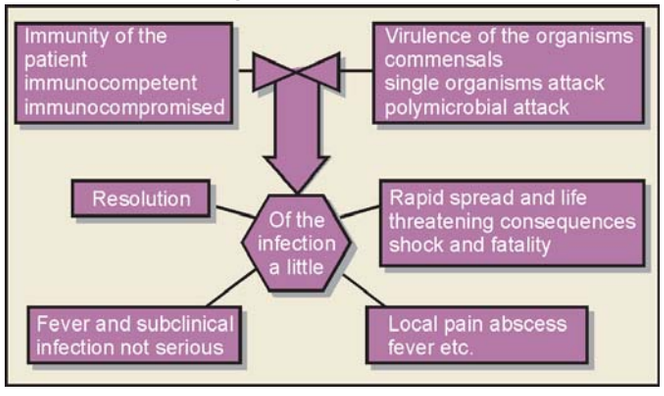


FIGURE 38.1: The Dynamic interaction of Host and the Organisms and the possible outcomes.

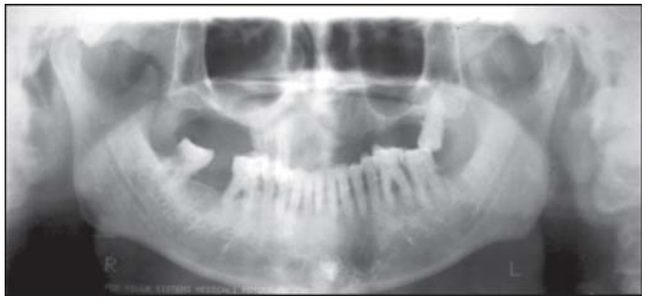


FIGURE 38.2: Showing the irregular radiolucency in the left premolar region suggestive of periodontal abscess. The clinical features of slightly raised feeling on the tooth, severe pain and fever will confirm the diagnosis (Kiran, Ajay N, and Bailoor DN 2003 Yenepoya Dental College and Hospital, Mangalore)

ORAL CANDIDIASIS

Candida albicans is a commensal organism in the oral cavity which has symbiotic metabolic interrelationship to *Lactobacillus acidophilus*. This organism belongs to the Cryptococcaceae family and exists in three forms.

a. Oval cells—blastophore form
b. Elongated cellular form— pseudohyphae form
c. Chlamydospore form—with a thick protective wall

Pseudo-hyphae form is the most commonly noted in the sheltered crevices of the oral cavity. Related species also seen occasionally are *C krusei*, *C guilliermondi*, *C psuedotropicalis* etc.

These organisms have very weak pathogenicity and hence, its presence should always alert the dentist to find what systemic or local problem is causing the candidal overgrowth rather than just treat the problem with antifungal therapy.

Predisposing Factors

1. HIV-infection
2. Severe malnutrition
3. Medications that may cause bone marrow depression like antityphoid therapy using Chloramphenicol
4. Patients receiving anti-cancer therapy by radiation or chemotherapy
5. Systemic antibiotic therapy
6. Systemic steroid therapy
7. Uncontrolled diabetes mellitus
8. Pregnancy
9. Sjögren's syndrome in elderly
10. Hypoparathyroidism
11. Rare syndromes like Di Georges syndrome etc.

Clinical Features

White soft curd like lesions which rub off leaving a raw bleeding surface. The rubbed off plaque contain fungal organism, desquamated epithelial cells, dead bacteria and fibrin. Large patches of such lesion when they are removed cause severe discomfort in form of burning or even dysphagia.

Persistent candidal infection converts to ultimately large erythematous areas, which have been labeled as atrophic candidiasis. Some authors have used the term antibiotic sore mouth to mean the same condition. Long-term broad-spectrum antibiotic treatment can cause these lesions.

Long-term persistence of the fungal infection can present in two forms:

i. Usually associated with dentures wearing termed as chronic atrophic candidiasis. This is typically seen on the palatal aspect of denture that is not fitting too well, patient who wears the denture through the night, or some who have systemic debilities.

ii. When the low irritation of the fungal hyphae becomes stimulated, oral mucosa is thrown into hyperplastic folds. We see hyperplastic white lesions of various sizes with characteristic curd covered appearance. Three sites show particularly typical lesions.
   a. *Angular cheilitis*—Linear lesions with cracked appearance and whitish borders at angle of mouth. Treatment should involve checking vertical dimension of the worn out denture, antifungal ointment and nutritional support.
   b. *Hyperplastic palatal lesions of Candida*— Range from asymptomatic to sever burning, usually respond to antifungal therapy locally.
   c. *Median rhomboid glossitis* at junction of anterior two-thirds and posterior one-third. Usually requires no treatment, but if burning is complained then antifungal cream may be used. (Candid ® ointment)

Persistent mucocutaneous types of candidiasis are usually associated with different endocrinopathies like hypo-parathyroidism, Addison's disease, or hypothyroidism.

Microscopically

Wooden spatula is used to scrape the suspected organism and then mixed with 20 percent potassium hydroxide and then the slide is smeared and examined for the hyphae in low power. Yeast elements may be stained using methenamine silver or PAS- periodic acid-schiff specially the psuedohyphae. In the chronic types there is significant association with epithelial hyperplasia in addition. Whether the close relationship between the Candida and hyperplasia is the cause or effect is not clearly established yet.

Treatment

Apart from determining which systemic problem is causing the *Candida* and focusing on it- local nystatin suspensions usually are effective for treating local lesions. Application of local Clotrimazole should continue at least five days after the complete remission of the lesions. Private practitioner should not attempt systemic antifungal therapy in the clinic since amphotericin B or ketoconazole given systemically will cause hepatotoxicity or bone marrow depression. Hospital referral is a better choice in chronic mucocutaneous syndromes.

PHARYNGITIS

Bacterial pharyngitis may be caused by Streptococci group A, non A, *Corynebacterium diphtheriae* and *Mycoplasma pneumoniae*. This infection is common in cold months and winters. Patients may present to a dentist with complaint of dysphagia, fever and local lymphadenopathy. The tongue is coated white with dots of red making it appear like "Strawberry tongue". In some children peritonsillar abscess called as quinsy may result. Oral penicillin, ampicillin, amoxycillin, etc., are drugs of choice in patients, with allergy erythromycin may be chosen. Paracetomol is the antipyretic of choice. Warm saline gargles and Hexidine® mouthwash helps to reduce the time of discomfort. Enterotoxin produced by *Staphylococcus aureus* gives two rare diseases, a) TSS (Toxic Shock Syndrome) and b) MLNS (Mucocutaneous Lymph Node Syndrome). Both of these are serious illnesses in which strawberry tongue is observed (Fig. 38.3).

FIGURE 38.3: Showing a 32-year-old male showing painful tonsillar swelling and problem of dysphagia. Fever lymphadenopathy and elevated WBC count resulted in a tentative diagnosis of tonsillar abscess. Surgical drainage and Oral Amoxycillin 500 mg tds × 6 days resulted in a reasonable relief. Pain control was obtained using Diclofenac Na (Divon ®) (Asha I, Girish R and Nagesh KS 2003, RV Dental College and Hospital, Bangalore, India)

TSS or Toxic Shock syndrome is caused by *Staphylococcus aureus* and some group A streptococci. It occurs in menstruating women who use intra vaginal tampons. Acute onset fever, vomiting, diarrhea, shock and low blood pressure are the main features seen in most patients. Red macular eruption on the skin and mucus membrane erythema are characteristic. Hemorrhagic dots are seen on the conjunctiva.

When TSS is diagnosed, prompt and immediate supportive therapy for shock is required. Intravenous antibiotics are recommended and ICU facility is a must.

MLNS OR MUCOCUTANEOUS LYMPH NODE SYNDROME

This disease was initially reported in Japan. Pre-school age children are commonly affected. Sudden rise in fever, bilateral conjunctival congestion, strawberry tongue, fissured lesions on the lips and reddish eruptions on palms and soles are some of the clinical presentations seen. Cervical lymphadenopathy is a persistent feature. Meningitis, arthritis and carditis can lead to fatality. • globulin is given for treatment and helps to reduce cardiac complications.

ORAL LESIONS CAUSED BY SEXUALLY TRANSMITTED DISEASES (STD)

Pharyngeal Gonorrhea

This infection of *Neisseria gonorrhoeae* occurs predominantly in sex workers (prostitutes) and homosexual men. The history of oral sex (Fellatio) is obtained in all the cases. Sore throat, fever and cervical lymphadenopathy are noted and most signs are of non-specific pharyngitis. Usually there is no need to culture specific organism and ciprofloxacin 500 mg orally for four days may be given.

Oral Lesions Caused by *Chlamydia trachomatis*

These organisms are strongly related with oro-genital exposure. Reiter's syndrome is strongly associated with this infection (80% cases in some series). This syndrome consists of urethritis, arthritis, stomatitis and eye inflammation. Studies also reveal that HLA-B_{27} haplotype is one of the risk factors. Tongue may show geographic areas of depapillation. These lesions are treated with doxycycline 100 mg twice a day for 7 days or 1 gm of azithromycin as a single dose therapy.

Syphilis

This is a chronic STD. It is specifically caused by *Treponema pallidum* and closely related species. If mother is affected during delivery, off spring may develop congenital syphilis through transplacental spread. Adults usually acquire it through heterosexual or bisexual activities and are termed as acquired syphilis. The acquired variety is divided into —

Primary Syphilis

After sexual act 2 to 4 weeks have to pass before itching and primary lesion appears on point of contact, on penis, labia of vagina, or lips and tongue. Primary chancre is the name given to this lesion, which is painless solitary, well defined and elevated.

Secondary Syphilis

This stage begins about 2 months after the primary chancre. Fever, lymphadenopathy and skin eruptions are noticed characteristically. One-third of the patients show mouth and throat lesions. These are called mucous patches. A typical mucous patch is macular linear and soon develops into slightly raised irregular ulcer which is covered with a grayish membrane. This is highly infectious stage and even an ordinary kiss can transmit the infection. In the scalp, patchy map like loss of hair may be evident and all over the body syphilitic leukoderma or areas of hypopigmentation may be seen. Serologic testing will demonstrate very high titers of antibodies.

Tertiary Syphilis

This stage is practically vanished with good management and routine use of antibiotics. It may appear anywhere in the body and are term mucocutaneous, osseous or neural etc. Painless nodules may appear all over the body and turn into gummatous lesions. Gummas have propensity to come on sternum, on the legs, the face and the scalp. In the oral cavity, hard palate, soft palate and tongue are the most common sites. In the palate, extensive scarring and perforation is noted.

Congenital Syphilis

These have been described as early and late lesions. The early lesions show bullous eruptions and mucous patches in the nasal mucosa, oral mucosa and the throat. The late findings which are quite rare today are the gummas located in the hard and soft palate leading to perforation with the problems of food regurgitation and difficulties of phonation. Lesions at angle of the mouth which are radiating in nature are termed as 'rhagades' and are permanent scars on the face of the person.

The residual effects of the damage done to the oral cavity have been described by many clinicians as the stigmata; the Hutchinsons teeth and Moon's (Mulberry) molars. Both are found on the permanent teeth due to persistence of the syphilitc infection on the tooth buds of the permanent teeth at birth. Literature shows two other findings that are variedly present, one is the Gothic palate or the high arched palate and the other is the protruding mandible which is due to the lack of development of the pre-maxilla region.

Treatment for syphilis has traditionally been the Penicillin Benzathine, Procaine and Oral but most strains becoming resistant, the clinicians today use Doxycycline 200 mg loading dose with 100 mg per day for two weeks. Erythromycin 500 mg qid at the dose of 2g per day for two weeks also has reported good results.

MYCOBACTERIAL INFECTIONS

Tuberculosis (TB)

In India it was believed two decades ago that by the turn of the century, TB will be eliminated due to extensive community work and education campaign. But it turned out to be just the opposite, today in 2001 as we see resurgence of HIV infection so do we observe the correlated increase in case of TB of different forms. The skin and oral manifestations of TB are varied and difficult to diagnose in the first clinical look even for the seasoned clinician. One of the ways to study and understand these findings is to classify them as per the route of attack of these organisms. The dental surgeons will see the more and more lesions on the neck and the oral cavity.

Exogenous source: When the organism is inoculated into a skin abrasion from TB patient to a healthy person, a papular lesion develops which turns into hard nodular area— this has been described as "Tuberculous chancre". Cold abscess and draining sinuses develop and the patient is forced to take treatment. Droplet infections of course result in the classical Pulmonary TB in which the regurgitation of sputum can result is a single shallow oral ulcer. Chest radiograph showing destructive lesion of the hilum and the scrapings of the floor of the ulcer showing the Acid Fast Bacilli AFB give strength to the diagnosis of TB ulcer of the oral cavity.

Endogenous source: Two major types of lesions seen in this variety are the Scrofuloderma and the Peri-orificial TB. The lymphadenopathy most commonly the cervical region shows multiple Lymph node involvement, this liquefies and then breaks down into draining sinuses and ulcerations.

Anogenital tuberculosis is the logical extension from the infection of the large intestine and may be the only manifestations in some patients.

Cutaneous Tuberculosis from Hematogenous Spread

When the immunity of the patient is fighting a loosing battle then these bacteria further spread by hematogenous route and termed "miliary tuberculosis". The nodules which are many and widespread are termed as ' Apple jelly' consistency on diascopic examination. Multiple oral lesions have also been frequently reported.

Dentists working for the hospitals are at increased risk for contracting TB. According to one of studies by Harlow RF and Rutkauskas JS [5] 1995, 132 hospital dentists were surveyed and five reported contracting TB from hospital through patient contact. Even the private dentists in practice have to be extra careful for the prevention of TB in endemic countries like India.

Dahmash NS et al (1995),[6] analyzed 80 patients between 60 and 88 years with pulmonary TB and Miliary TB. Chest X-ray was diagnostic in 71.2%, the organism were detected in sputum specimens of 62.5%,and flexible fibreoptic bronchoscopy (FOB) revealed that it was a very useful diagnostic tool for detection of AFB- acid fast bacilli.

In this series the overall mortality reported was 21%. This is not very surprising considering the fact that most patients were above 70 years of age.

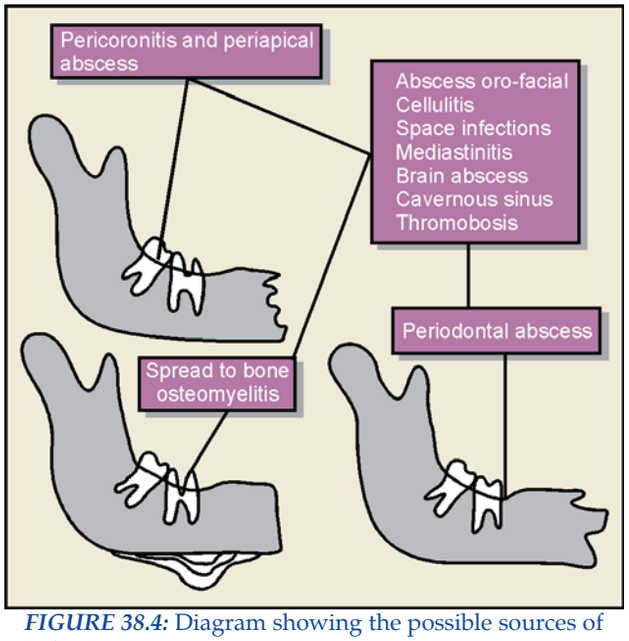


FIGURE 38.4: Diagram showing the possible sources of infection in the oral cavity and its tentative target areas

In the oral cavity TB may present additionally as a large non-healing ulcer. Histopathology is non- specific.

Mycobacteria Leprosy

The worldwide prevalence of leprosy is reported to be 5.5 million cases. The majority of affected persons live in the tropics and subtropics. Worldwide, 80% of the cases are found in 5 countries: India, Myanmar, Indonesia, Brazil, and Nigeria.[7]

The Hansen's disease is the specific infection caused by *Mycobacterium leprae*. It is widespread in India, South east Asia, Burma, Central Africa, Middle east, and central and south America. Most clinician believe that the transmission is from droplet infection. The incubation period is between 3 and 5 years for leprosy and some studies have shown that some individuals are more prone to get Hansen's due to some quirk in their immune mechanisms.

It starts as hypo or hyper-pigmented patch on face, extremities, or trunk. The sensation in these irregular patches is diminished to gone. Hansen's is further characterized by loss of eyebrows eyelashes and body hair and sensory loss of the extremities. Facial skin becomes thickened and thrown into folds and the destruction of the nasal septum and the anterior nasal spine results in the misshapen nose. Tongue may show nodular infiltrations and the gums may also be involved.

Special acid-fast staining methods (e.g. Wade-Fite) must be used to identify *M leprae*, since the organism may lose its acid-fast characteristic when fixed and stained by the conventional Ziehl-Neelson technique. Communication with the pathologist about the possibility of Hansen's disease should facilitate diagnosis.

Animal reservoirs of leprosy have been found in 3 species: 9-banded armadillos, chimpanzees, and mangabey monkeys.

Odontogenic infection starts as slow growing swelling, which is warm and irritates with a dull pain. Most patients feel feverish and pains radiate to adjacent regions. There are some terms that we need to clarify before we discuss what happens in an odontogenic infection. These terms are Cellulitis, Abscess, Draining Sinus, Antibioma and space infection.

Periodontal Abscess

Acculmulation of the pus in the periodontal ligament space is usually seen in medically compromised patients and it can become a source for spread of infection and lead to cellulitis, space infection or focus of infection to distant anatomic regions (Fig. 38.5).

FIGURE 38.5: Showing 43-year-old male patient with uncontrolled diabetes mellitus with radiographic confirmation of the clinically suspected periodontal abscess resulting in the submandibular and buccal space infection. (Prasanna K, Nillofer S, Bailoor DN 2003, Yenepoya Dental College and Hospital, Mangalore).

Cellulitis

It is a first stage of the spread of infection from periapical or periodontal lesion. Diffuse inflammatory response warm swelling that is firm to hard in consistency and tender to palpation.

This could convert into a well localized walled off, lesion or it could become a rapidly spreading infection which crosses the potential anatomical spaces and follows the fascial planes termed as Space infection.

Abscess

It is an localized collection of pus, which is warm, soft and fluctuant and soon becomes pointed and drains either intra-orally or extra-orally. Dull to severe pain prior to draining.

Draining Sinus

It is an abscess which has developed as well epithelialized tract through which the pus drains periodically since the cause of the infection has not been treated. Most draining sinuses are not painful but give a very foul odor in the oral cavity.

Antibioma

When a patient with odontogenic abscess indulges in self medication with over the counter antibiotics, the abscess instead of becoming pointing and then draining, it gets organized and fibrous. The patient has a tough fibrous swelling, which has dull pain and sporadically causes fever and constitutional symptoms. Once antibioma sets in, it takes a long time to resolve even after the cause of it is treated, sometimes as long as 3 to 4 months. If it is causing an esthetic defect then it must be surgically excised.

Space infection spreads through fascial planes and can present as typical swellings of the face and some if not treated promptly result in fatality. We shall discuss the following space infections briefly.

- Buccal space infection
- Masseteric space infection
- Infra-orbital space infection
- Ludwigs angina

Other Types of Infections

Cavernous sinus infection, Infective sialoadenitis and infective arthritis also need to be discussed in this conjunction.

- **Buccal space infection:** The periapical lesions of mandibular or maxillary molars frequently spread laterally through the cortical plate into buccinator muscle through the posterior insertion of this muscle. Pain, redness and swelling of the cheek region of the affected side is usually prominent. Fever is a regular feature.
- **Masseteric space infection:** When the odontogenic infection spreads posteriorly between the ramus and potential space under the masseter, it results in a firm, non fluctuant, tenderness at the angle of the mandible. Spasm of the masseter could result in the trismus (difficulty in mouth opening).
- **Infra-orbital space infection:** It is an microbial attack that spreads superiorly from the maxillary teeth anterior to the first molar region. Organism spread lateral to the nasal region superficially on the facial aspect and may cause enroachment of the eyelids and blurring of vision.
- The Ophthalmic angular viens lack valves and these may facilitate the spread of virulent organism right into the brain and cause Cavernous sinus thrombosis.
- **Ludwigs angina:** When a severe infection from the mandibular teeth rapidly spreads to the submandi-

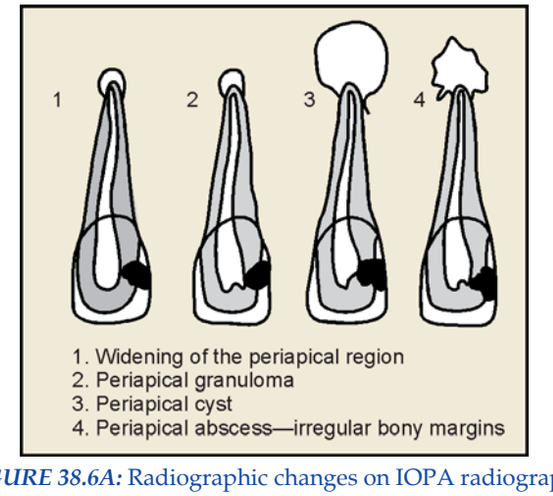


FIGURE 38.6A: Radiographic changes on IOPA radiograph

bular, submental and sublingual spaces bilaterally we have a potentially life threatening situation which causes the tongue to rise superiorly and fall back, and airway obstruction may require immediate tracheostomy apart from r treatment.

When patients suffer from partial to complete Xerostomia the ascending infection through the salivary glands ducts can cause infective sialoadenitis. The suppurative attack on any of the major salivary gland is very painful and it is very recalcitrant to long term antibiotics, and in many cases the gland itself needs to be surgically excised to give relief.

- Ascending infection from the ramus of the mandible, middle ear infection or mastoiditis can result in the infection into the TMJ which needs to be aggressively treated or else the fibrous and subsequent bony ankylosis can result (Fig. 38.4).

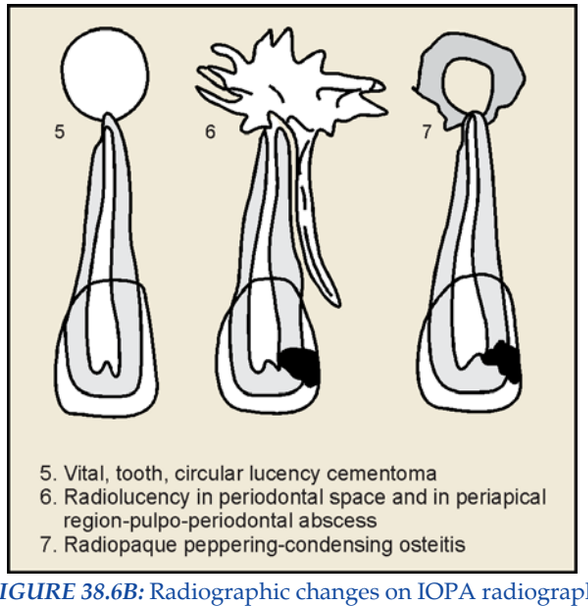


FIGURE 38.6B: Radiographic changes on IOPA radiograph

Four steps involve the treatment of any of the odontogenic infection—

- Remove the source
- Drain the pus
- Give suitable analgesics and antibiotics as required
- Supportive care of nutrition and nursing.

The infection from the pulp or periodontal ligament could reach the periapical region and cause —

1. **Acute periapical abscess—** Shows widening of periodontal ligament space.
2. **Periapical granuloma—** Small radiolucency with sharp demarcated borders
3. **Chronic periapical abscess—** Irregular radiolucency with hazy borders
4. **Periapical cyst** (Radicular cyst)— Well demarcated reactive border-circular radiolucency
5. **Sclerosing osteitis—** Radiopaque irregular areas trying to wall off the infection
6. Go further into the jaws and cause—Acute or Chronic osteomylitis**.**
7. Rarely spread through the blood vessels and cause wide spread hematogenous spread (Figs 38.6A and B).

1, 2 and 3 in the above are painful but 4 (periapical cyst) is usually painless on vertical percussion unless secondarily infected.

In all the above conditions the tooth is non-vital to Electronic pulp testing.

Root canal treatment is usually a standard procedure for treatment for all the above conditions to establish drainage and do further treatment.

5. Single or multiple radiolucencies with vital teeth, totally asymptomatic and revealed on routine radiographic examination usually cementoma is the diagnostic label used. It goes through partial radiopacity to total radiopacity in 2 to 3 years time.

6. The widening of periodontal ligament space on either the mesial or the distal aspect that is continous with the periapical region could be diagnosed as pulpo-periodontal abscess—here both endodontic and periodontic therapy is indicated.

7. When the infection is low grade and the vitality of the bone is high (young individuals) there is radiopaque peppering of osteoblastic reaction around the radiolucency which is suggestive of the walling off effect. Prognosis for such teeth is very good.

Osteomyelitis of the Jaws

Any of the above sources of infection may spread to the jaws and cause the infection of the bone. When the infection affects the marrow and the periosteal region and spreads in between the two cortical plates it truly is termed as

Osteomyelitis. Within the first ten days of the infection it is termed as Acute Osteomyelitis and no bony changes are seen. Severe bone pain and some areas of intra-oral drainage of pus may be the only visible complaints of the patient (Figs 38.7 and 38.8).

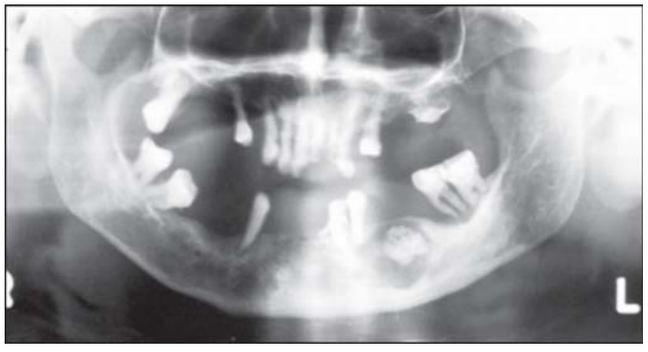
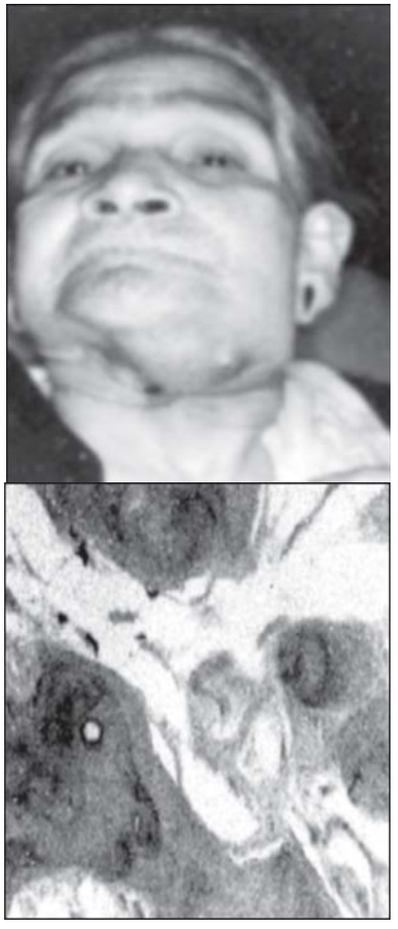
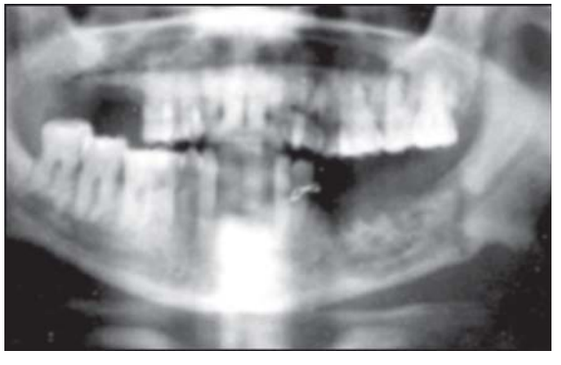


FIGURE 38.7: Showing a 49 year old male with severe pain in the left side of face in the mental foramen region with paresthesia of the lip. Radiographically a radiopaque sequestrum is seen and it is surrounded by a radiolucent zone. Chronic osteomyelitis was treated with sequestrectomy and antibiotics for twelve days (Courtesy of Mahima Patil and Kartikeya Patil JSS Mysore, Mysore 2004)

FIGURE 38.8: Showing the irregular bone destruction and radiopaque sequesteration seen. Bone pain and paresthesia made clinicians suspect chronic osteomyelitis. Patient was a known diabetic under some alternative medicine treatment (Courtesy: Umarji H, Ani John GDC Mumbai 2003)





FIGURES 38.9A to C: Showing a 58-year-old lady with draining sinus of six months duration. She was a tribal and did not have proper access to health care. Severe bone pain, paresthesia of the left lip and angle of mouth region and moth eaten appearance on the OPG radiograph revealed that Chronic Osteomyelitis was to be treated (Courtesy: Sripathi Rao BH, Bailoor DN, Yenepoya Dental college and hospital, Mangalore 2002)

Always investigate for:

- Systemic compromise in form of bone marrow depression
- Diabetes mellitus
- Long term steroid therapy
- End stage renal disease
- Malnutrition
- Anemias
- Paget's disease
- Fibrous dysplasia
- HIV
- Irradiation for cancer and any other causes that depress the immunity of the patient.

Chronic Osteomyelitis (COM) presents in quite a few clinical appearances. It is common in mandible and most series report male predominance (Fig. 38.9).

Patient reports severe bone pain, tender lymphadenopathy, lack of sensation in the some parts of the face (paresthesia), depending on site of the Osteomyelitis. Fever and constitutional symptoms predominate in chronic osteomyelitis. Radiographically we see moth eaten appearance the jagged radioluceny, some areas of radiopacity of dead bone termed as sequestrum, some light radiopacities at the edge of radiolucency suggestive of new bone formation usually called the involucrum. Clear punched out radiolucencies indicate the region from the place where the pus may drain out and are termed as cloacae.

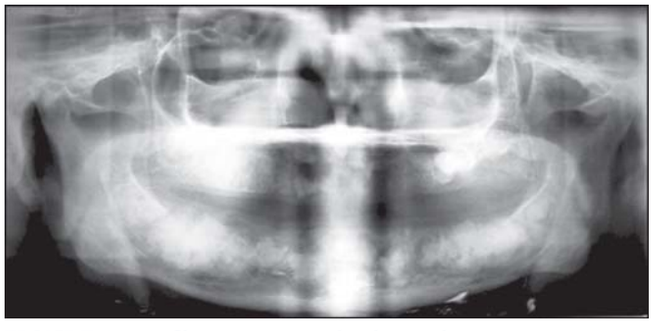


FIGURE 38.10: Showing irregular bone deposition in entire mandible and parts of maxilla. Dull pain and recent complete dental extraction was recorded in the dental history. The cause for the chronic sclerosing osteomyelitis was under investigation (Courtesy: Mahima Patil, Kartikeya Patil JSS Mysore, Mysore 2004)

Diffuse-sclerosing Osteomyelitis (DSO)

This is characterized by diffuse bony hard swelling with dull pain, raised ESR, low grade fever and tingling and paresthesia of some parts of the face. Radiographic patterns are characteristic. Radiopaque and radiolucent regions interspersed like whorls of clouds and usually large areas of jaw may be involved (mandible mostly). It affects one side and rarely crosses midline. This criss-crossing of radiopaque and radiolucent represents the attempted repair of bone to low grade osteomyelitis infection. Many of the research workers have outlined the diagnostic benefits of Scintigraphy using Tc^{99m} polyphosphate. There is a distinct uptake of the radionuclide isotope in the diseased parts of the bone (Fig. 38.10).

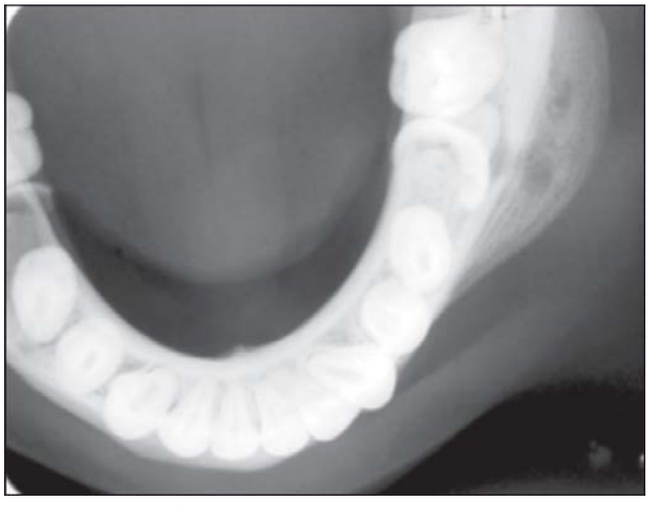


FIGURE 38.11: Showing 13 year old with dental infection presenting with bony hard swelling, pain ful with pyrexia and lymphadenopathy. Occlussal radiograph typically shows the Garres Osteomyelitis with its onion peel appearance (Courtesy: Kartikeya Patil, Mahima Patil JSS Mysore, 2004)

Proliferative Periostitis (Garre's Osteomyelitis) PP

This type of bone infection is seen in the children and young adults.The low grade infection trickles down to the sub-periosteal region and stimulates the creation of new bone in onion peel like appearance. Patient presents with either painless or dull pain with bony hard knob like swelling. As weeks proceed the classical signs of either DSO or COM develop and then all the symptoms begin to appear (Fig. 38.11).

All the osteomyelitis patients require bactericidal antibiotics from two to four weeks, surgical decortication and removal of any dead bone (sequestrum). In difficult cases hyperbaric oxygen has been used since a major part of the infection is anerobic in nature.

Detailed clinical pharmacology of antibiotics, analgesics and corticosteroids has been discussed in the Chapters 35 and 36.

Management of Infections

- In apparently immunocompetent individuals
- In immunocompromised individuals
- In patients with other systemic disorders

The spread of infection locally to the orofacial region can be from a periapical abscess, periodontal abscess, pericoronal abscess or rarely secondarily infected major ulceration. Hematogenous spread from different parts of the body remain a veritable possibility.

In immunocompetent individuals if an periapical abscess is detected then draining it through the root canal and follow up by NSAIDs like Ibuprofen (Brufen® 400 mg three times a day for three days) is usually sufficient. Routinely there is no need for antibiotic prescription. There is a general over-prescription of antibiotics by dental practitioners, which may be one, the causes for a lot of antibiotic resistance that we see in infection management. However, if the patient has systemic compromise like diabetes mellitus or bacterial endocarditis then the recommended prophylactic bactericidal antibiotics plus those recommended for the local suppuration must be prescribed.

Usually first line antibiotics used by dentists today are the oral penicillins, Amoxycillin, Amoxycillin with clavulanic acid combinations, Amoxycillin with *Lactobacillus* spores. In case of non-response/ allergic limitations, macrolides are the drugs of choice. Drug dosages and new formulations are coming in everyday. Dentist must refer a good manufacturers index and consider even the pricing aspect of the different antibiotics. Since many of our patients are poor. New drugs should be prescribed only after looking at the evidence available on the Internet regarding the side effects from peer-reviewed sites. Pallasch TJ (1997)[4] support the use of Erythromycin as the most effective drug for many orofacial infections.

The newer macrolides, azithromycin and clarithromycin, should also prove. They have the advantages over erythromycin of less GI toxicity, higher tissue concentrations, greater gram-negative spectrum, and once or twice daily dosing for better patient compliance. Macrolide concentration in inflammatory cells and transport to the site of infection is a distinct advantage over other antibiotics.

Sandor GK et al (1998)[1] mentions that in mixed orodental infections the Clindamycin is the first-line of treatment. He reasons that Penicillins have become ineffective due to increasing levels of resistance. Most acute orofacial infections are of odontogenic origin. In normal hosts, however, they usually do not occur without some type of predisposing condition. Early recognition and management of acute orofacial infections is critical, because rapid systemic involvement can occur, especially in children. Antimicrobial therapy has an essential role in the management of these infections. If it is initiated before surgery, it can shorten the period of infection and minimize associated risks. The etiology of odontogenic infections is usually attributed to the endogenous flora of the mouth, and not to the introduction of non-resident bacteria. Odontogenic infections are typically polymicrobial; however, anaerobes generally outnumber aerobes by at least four fold. The penicillins have historically been used as the first-line therapy in these cases, but increasing rates of resistance have lowered their usefulness. Bacterial resistance to this class of agents is predominately achieved through the production of beta-lactamases. Clindamycin, because of its broad spectrum of activity and resistance to beta-lactamase degradation, is an attractive first-line therapy in the treatment of odontogenic infections.

Role of anti-inflammatory drugs and Corticosteroids during routine dentoalveolar surgery (Table 38.1).

Commonly preferred anti-inflammatory drugs are Ibuprofen and Ibuprofen with paracetomol combinations (Brucet DT®, Imol®). Alexander RE and Throndson RR (2000)[3] mention that only when significant surgical trauma is inflicted or the patient is judged to be at risk for excessive edema then the steroids should be used. They should be started prior to surgery, night before and the long acting ones preferred over the short acting ones. Most of these recommendations are empiric and we must await more evidence based information in the coming years.

Table 38.1: Showing empirical recommendations for use of corticosteroids for perioperative dentoalveolar surgery—Alexander and Throndson (2000)[3]

Type of drugs used	*Route*	*Dosage night before*	*Dosage day of surgery*	*Post op.*
Dexamethasone	Oral	4-8 mg	8-16 mg	4 mg after 8 hr
Methyl prednisolone acetate (MPA)	IM	8-16 mg tablets	40 mg IM	Nothing
Methyl prednisolone sodium succinate (MPSS)	IV	Nothing	125 mg IV drip 8 mg tablet of MPSS	Every 6 hr for 4 doses

CONCLUSION

Infections of orofacial regions are common in dental practice and dentist must use healthy commonsense in prescriptions of antibiotics. *Never use a sledge hammer to kill an ant!*

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39 Radiotherapy in Head and Neck Cancers

DEFINITION

Radiotherapy, also called radiation therapy, is the treatment of diseases using ionizing radiation. (Concise Oxford Dictionary). Ionizing radiation is used for both therapeutic and diagnostic purposes. For therapy high-energy radiation in megavoltage range is preferred where as for diagnosis kilovoltage energy is used.

PRINCIPLES OF RADIOTHERAPY (RT)

The radiotherapy is based on the basic principle that rapidly proliferating cells are more sensitive to ionizing radiation compared to normal cell. This differential cell kill is used for the treatment of tumors.

Radiobiology

Ionizing radiation when passes through the tissue of a patient affects the biology of both normal and tumor tissues. This radiation causes both direct and indirect effects on biologic targets. The DNA of a cell may be directly affected by the secondary electrons generated as ionizing radiation interacts with tissue. The radiation may also have an indirect effect due to the formation of free radicals; these free radicals in turn cause mostly of the chemical damage to the DNA. In addition, there are number of other cellular functions that are disrupted by radiation induced damage. This damage is modified by oxygen concentration, temperature and other intracellular components.

Goal of radiotherapy is to sterilize tumor and to preserve adjacent normal tissue. Ionizing radiation deposits energy that injures or destroys cells by damaging their genetic material, making it impossible for these cells to continue to grow.[1]

- Lethal dose for normal and abnormal tissues is about the same
- Normal tissues have greater ability to repair sublethal damage between doses of radiation than neoplastic cells.

The effects produced depend on factors like Oxygen Enhancement Ratio (OER), Linear Energy Transfer (LET), and Relative Biologic Effectiveness (RBE).

OER is the ratio of doses of radiation required to produce a given amount of damage without and with oxygen.

RBE is the ratio of dose of 250 kV X-rays to that of test radiation required to produce equal amount of biologic effect.

LET is the energy transferred by the radiation per unit length of track.

Response of cells to radiation depends upon several factors such as radiation quality, dose rate, dose fractionation, oxygen tension, cell stage, presence of chemical

protectors and sensitizers, recovery and repair process. Presence of oxygen is among the best known sensitisers of radiation damage (Fig. 39.1). High LET radiations like neutrons and protons cause more radiation damage compared to low LET radiations like X-rays or gamma rays. Cells in S-phase generally are more radioresistant as compared to cells in G2 and M phases.

PHYSICAL CONCEPTS IN RADIATION ONCOLOGY

Ionizing radiation used to treat cancers is divided into electromagnetic and particulate components. Electromagnetic radiation is the predominant therapeutic modality for clinical radiation therapy. Clinically significant particulate radiations include electrons and to a small but important degree neutrons and protons. The electromagnetic spectrum ranges from low energy to higher energy. It is the high-energy spectrum where gamma rays and X-rays are used to obtain the results desired in radiation therapy.

X-rays and gamma rays are essentially the same type of electromagnetic radiation (photon). They differ in the ways they are produced. X-ray are produced by man made devices by slowing down fast moving electrons. Gamma rays are emitted from a radioactive isotope as part of the process of naturally occurring radioactive decay. RT with particulate radiation differs from photon radiotherapy in that it involves the use of fast-moving subatomic particles to treat localized cancers. Most particles (neutrons, protons etc) deposit more energy along the path due to their mass, thus causing more damage to the cells they hit. They are called high LET radiation. Recent advance in radiotherapy research is the use of radiolabeled antibodies to deliver doses of radiation directly to the cancer site (radioimmunotherapy). Tumor-specific antibodies from certain tumor cells are attached to radioactive substances (radiolabeling) and injected into the body, which actively seek out the cancer cells and destroy them by the cytotoxic action of the radiation. This approach can minimize the risk of radiation damage to healthy cells.

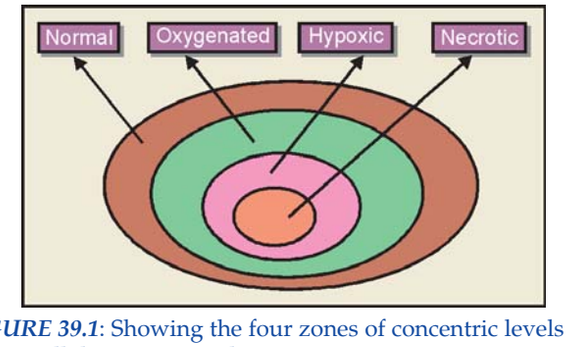


FIGURE 39.1: Showing the four zones of concentric levels of cell destruction and sensitivity in a tumor mass

METHODS OF DELIVERY OF RADIOTHERAPY

Based on the method of delivery of radiation the radiotherapy treatment is classified into teletherapy, brachytherapy and internal therapy.

Teletherapy

Radiation given using machines kept at a distance away from the patient.

Superficial Therapy

Superficial voltage machine generate X-rays 30 –125 KV. This is used to treat skin tumors.

Orthovoltage Therapy (Kilovoltage)

Orthovoltage machines produce medium energy X-rays in the range of 200-300 kv. Primarily used to treat superficially situated tumors. In this situation skin dose is high and cannot be used to treat deep seated tumors.

Telecobalt Teletherapy (Co^{60})

i. Emits gamma rays, with average energy 1.25 Mev
ii. Easier and safer to irradiate deeply seated tumors
iii. Maximum dose occurs 5 mm below skin surface; this "skin sparing" effect results in fewer skin reactions

Linear Accelerator

These machines produce X-rays above 1 MV and are referred to as megavoltage machines (Fig. 39.2). These are the most commonly used radiotherapy machines.They are used to treat deep seated tumours without producing skin reactions (skin sparing effect).

FIGURE 39.2: Linear accelerator which produce 8 and 15 MV photons and electron energies ranging from 4-18 MeV (Courtesy: Ramdas K RCC Trivandrum 2004)

Brachytherapy

Radiation source is kept in close contact or with in the tumour. The commonly used isotopes are Radium^{226}, Cesium^{137}, Iridium^{192} and Iodine^{125}. The treatment usually lasts for few days.

Brachytherapy is often divided into three types:

a. *Intracavitary brachytherapy*: Radioactive isotopes are kept inside a body cavity, e.g. carcinoma of nasopharynx and carcinoma cervix
b. *Interstitial brachytherapy*: Radioactive isotopes are implanted into the tumor, e.g. Carcinoma tongue and buccal mucosa (Figs 39.3 and 39.4)
c. *Mould therapy*: Radioactive isotopes are kept in close contact with the tumor, e.g. carcinoma of hard palate and skin cancer

Internal Therapy

Radioisotope is either injected or taken as a drink to treat tumors. For example: Radioactive iodine (I^{131}) in the treatment of thyroid cancer.

Phosphorus –32 in the treatment of polycythemia vera.

External Radiotherapy

External radiotherapy is normally given as a series of short daily treatments in the radiotherapy department using teletherapy machine (telecobalt/linear accelerator). Preparation for radiotherapy often takes longer than the actual treatment itself pinpointing accurately the position of the cancer with the help of simulator and/or computed tomography (CT) scans, which give a three-dimensional picture of internal organs. In planning the radiotherapy, the size of the cancer, its sensitivity to radiation and the sensitivity of the surrounding tissues are taken into account. The area to be irradiated is marked, often by tiny tattooed dots on the skin. In head and neck cancers, a mould or shell is often made to immobilize the treatment area and also to localize the radiation area. If patient is anaemic blood transfusion may be given to increase the amount of oxygen in the blood, since this has been found to make some cancers more sensitive to radiotherapy.

Steps in External Beam Therapy (EBT) Procedure

A. ***Mould and mould room:*** For head and neck radiation a mould is made for the head and neck region to immobilize the treatment part and to mark the radiation field.
B. ***Simulation:*** An X-ray unit, which has all the mechanical functions of a therapy machine, is used for this.
C. ***Planning:*** The time between simulation and starting of treatment is used to plan the treatment using the data collected in the simulator and from the CT scan, which is transferred to the planning computer where appropriate calculations are done. This ensures the attainment of the prescribed radiation dose to the tumors cells while the dose to normal tissue is kept to a minimum. During this process the patient is positioned as for the treatment and the site of treatment is clearly identified and marked on the mould or tattooed on the patients body.

Treatment: Blocks and shields made of lead ensure that radiation reaches only the target tissue. Port films are X-ray pictures taken during treatment to ensure the precision of targeting and so is 3-D computer software. Shielding is mandatory for following organs after receiving the tolerance dose. Brain stem - above 54 Gy; spinal cord - above 45 Gy; Mandible and TMJ above -70 Gy; temporal lobe above 60 Gy.

SIDE EFFECTS OF RADIATION

Radiotherapy side effects are classified into acute and late. Acute reaction occurs during and immediately after treatment. It is self-limiting. Acute effects are related to dose per treatment, total dose, volume of tissue irradiated and the site. These reactions are limited to the area irradiated and not seen outside the treatment volume. This is mainly due to the inflammation of the tissues during treatment. Symptoms include xerostomia, pain in the mouth and throat, falling of hair within the volume and skin reactions. During therapy salivary secretion decreases and it become thicker and forms a coating over the tongue. This cause change in the pH of the saliva, which can lead to changes in the bacteria flora. During radiotherapy there will be alteration of taste. The mucous membrane gets inflamed leading to patchy ulceration. There can be superadded bacterial and fungal infections. This is managed by antibiotic, antifungal and analgesics. During therapy patients are advised to take high calorie non-spicy bland food. Frequent use of soda bicarbonate and saline mouthwash is advisable. They should not use very hot and cold food. Application of creams and oils and rubbing of the treatment area with rough clothes should be avoided.

Late Reaction

This is the dose limiting toxicity and usually occurs months or years after treatment. This depends upon the dose per fraction, total dose and volume of tissue irradiated. This includes necrosis, fibrosis, fistula formation, non-healing ulceration and damage to specific organs such as spinal cord transection and blindness. Since late sequelae often are progressive and treatment is ineffective, they must be anticipated and avoided or minimized whenever possible using good treatment technique.

ADVANTAGES OF RADIOTHERAPY

- No tissue or functional loss
- Good cosmetic outcome compared to surgery
- Control of subclinical disease in the regional nodes is possible without added morbidity
- Can simultaneously treat multiple primaries.
- Better surgical salvage of radiotherapy failures than radiotherapy salvage of surgical failures.
- Rare treatment related mortality

DISADVANTAGES OF RADIOTHERAPY

- Undesirable acute side effects such as painful mucositis, loss of taste, dryness of mouth etc.
- Potential late complications of soft tissues and bone
- Development of second malignancy
- Protracted treatment course
- Requires good infrastructures

FRACTIONATION IN RADIOTHERAPY

Fractionation is a term used to describe the manner in which daily dose of radiation is given. Fractionation of the total dose of radiation helps in minimizing normal tissue reaction. The clinical effects of fractionated radiotherapy are influenced by the ability to repair sublethal damage, reoxygenation of tumour during the course of radiation, repopulation of tumour and normal tissues between fractions and redistribution of cells into a more sensitive phase in cell cycle treatment. (The 4 R's of radiobiology)

Conventional Fractionation

Conventional fractionation is the application of daily doses of 180-200 cGy and 5 fractions per week to a total dose of 40-70 Gy depending upon the type of tumor.[2]

Hyperfractionation

Two or more fractions per day of reduced dose (115-120 cGy) with overall treatment time similar to that of conventional fractionation..Hyperfractionation helps in increasing the total dose without increasing the late reactions.

Accelerated Fractionation

Accelerated fractionation is a means of decreasing the overall duration of treatment in an effort to reduce the repopulation of tumor cells in rapidly proliferating cancers. Repopulation (tumor-cell regeneration) occur during treatment when the overall duration of treatment is

increased.Shortening of overall treatment time can increase the tumor control in selected situation.

Accelerated Hyperfractionation

Delivering two or more fractions per day of normal dose per fraction helps in reducing overall treatment time without increasing the risk of late complication.

Concomitant-boost Technique

A variant of accelerated fractionation is the concomitant-boost technique. With this technique, treatment is delivered once daily for the first 3.5 weeks and then twice daily during the final 2 to 2.5 weeks, when tumor cells can begin to repopulate more rapidly.

Hypofractionation

Here less than four fractions per week with higher dose per fraction than conventional is planned. In selected situations this is found to be useful especially in the treatment of melanomas.

Split Course Therapy

Radiation is given in small courses with a rest period in between.

BRACHYTHERAPY

The term brachytherapy was first proposed by Dr.G. Forsell in 1931.

It is derived from the Greek word *brachio*, meaning short, and refers to "treatment with a radioisotope at a short distance less than 5 cm from a tumor. Interest in brachytherapy remained low due to the radiation exposure to operating staff.The introduction, in the early 1960s, of megavoltage linear accelerators capable of producing improved teletherapy dose distributions further slowed the use of brachytherapy. The development of safe after loading technique especially the high dose rate remote after loading methods has given renewed interest in this field. The advantage of brachytherapy is that it is a highly localized form of treatment causing minimal and excellent tumor control. Short treatment time is another advantage of this technique. The disadvantage is that it can be used only in selected cases especially in early stage disease at accessible sites. It requires anesthesia and excellent expertise. This is an invasive procedure.

Brachytherapy may be used either alone or in combination with external beam radiation and/or surgery. The radioactive sources used for brachytherapy come in the form of small seeds, needles or wires. The dose of radiation and length of time prescribed will depend on the tumor size, location, and sensitivity to radiation. Generally a dose of 65 Gy over 6-7 days is given when it is used as the sole treatment.

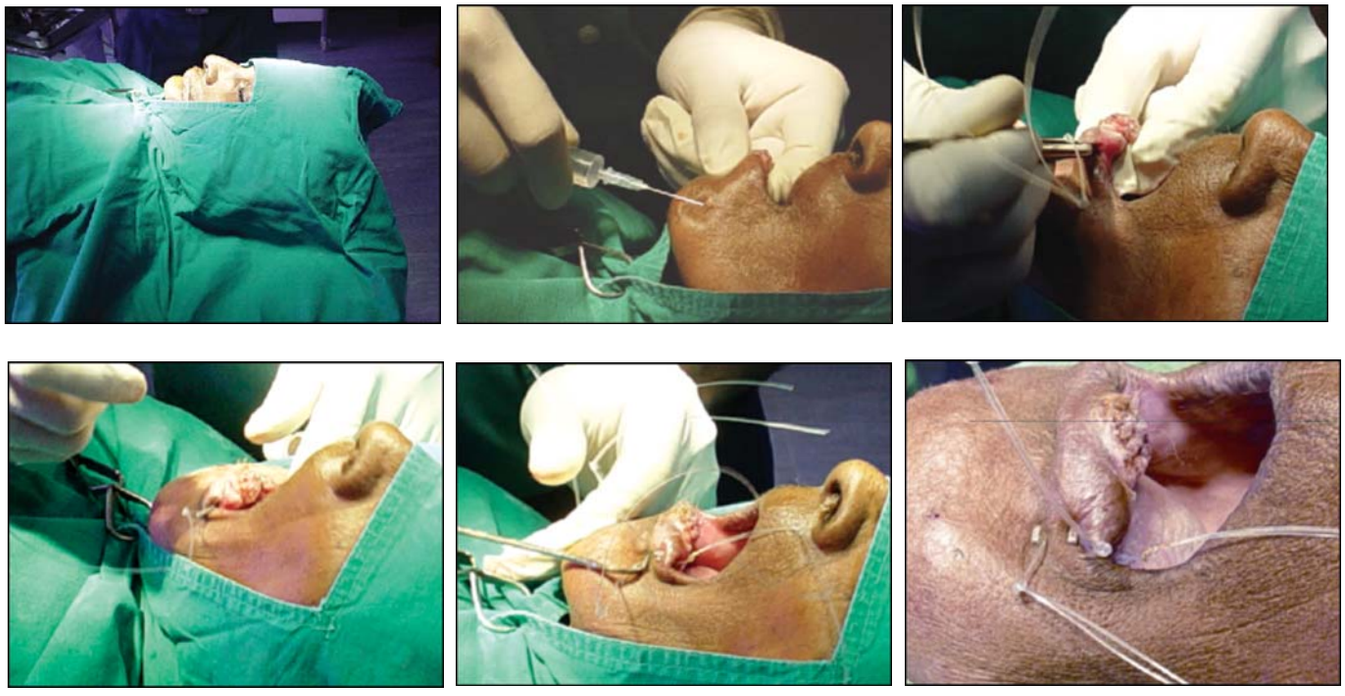
Depending upon the loading brachytherapy is classified into preload and afterload techniques. In preload technique radioactive isotope is directly handled by the staff and all staff members involved in the procedure get radiation whereas in after load technique first metal or plastic tubes are inserted into the tumor and these tubes are later loaded with radioactive source. This can be done manually or with the help of machine (remote after loading techniques). In remote after loading technique the radiation hazard is almost nil.

High Dose Rate (HDR) Brachytherapy

This is an outpatient form of radiation treatment that has become very popular in recent years. A small machine containing a high dose rate radioactive isotope is used for this. The source is transferred into the tubes kept within the tumor using a catheters attached to the machine. After the treatment the source goes back to the machine. All the source movements are controlled with the help of computer.

MANAGEMENT OF HEAD AND NECK CANCER

Head and neck cancer is a locoregional disease and majority of these patients die because of uncontrolled loco-regional disease. Distant metastasis is extremely rare in these tumors. Therefore local forms of treatment like surgery and radiotherapy either alone or in combination plays an important role in the management of these cancers. Chemotherapy alone has no role for curative treatment. Chemotherapy is used in recurrent or residual diseases for palliation of symptoms. In curative settings it is used along with surgery and/or radiotherapy.



FIGURES 39.3A to F: Brachytherapy procedure of carcinoma lip

FIGURE 39.4: Patient having carcinoma Upperlip undergoing HDR brachytherapy using microsselectron (Courtesy: Ramdas K RCC Trivandrum 2004)

The choice of treatment depends on factors such as cell type or degree of differentiation, site and extent of the primary lesions, gross characteristics of the tumor (exophytic, superficial vs endophytic, infiltrative), involvement of bone and muscle, metastatic nodal status, likelihood of complete surgical resection, possibility of preservation of speech or swallowing mechanisms, physical condition, social status and occupation of the patient, experience and skill of both the surgeon and the radiotherapist, infrastructure, cooperation and wishes of the patient.

Baseline Investigations

- Complete history and physical examination: Height, weight, performance status and nutritional status
- Complete diagrammatic and descriptive documentation including measurements of the extent of primary and regional disease
- ENT evaluation of the head and neck region including endoscopic evaluation.
- Routine blood tests like TC, DC, BUN, serum creatinine and blood sugar estimation.
- Chest X-ray, orthopantomogram and X-rays of the affected site.
- High resolution CT/MRI of the primary site and the neck optional
- Nodes not palpable and found only on CT or MRI scans: Size with imaging must be =1.0 cm in its minimal axial diameter/contain necrotic regions regardless of size
- Biopsy from the primary site. If no primary is detected, FNAC from the regional node.

- Baseline quality of life (QOL) assessment (prior to the start of protocol treatment) and pregnancy test for women of childbearing potential is also advocated.

Tumor Board

Couple of decades ago the specialist who saw the cancer case would end up treating it. If it happened to be a radiotherapist the patient would get radiotherapy and if he was a surgeon he would get operated immediately etc. to prevent this most hospital authorities and senior oncologists have recommended formation of tumor boards. The team includes:

a. Oral medicine specialist
b. ENT- oto-rhinolaryngologist
c. General surgeon
d. Radiotherapist
e. Chemotherapist
f. Immunotherapist
g. Registered nurse with onco-training
h. Clinical psychologist
i. Dietician
j. Religious workers or priests according to the religious belief of the patient.
k. Additional specialist if required.

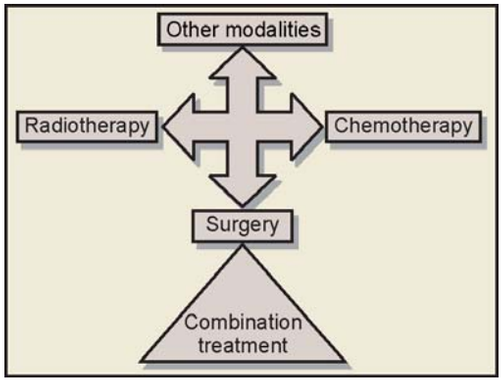


FIGURE 39.5: Showing the different modalities of treatment that the tumor board selects for the cancer patients (Ramdas K 2004)

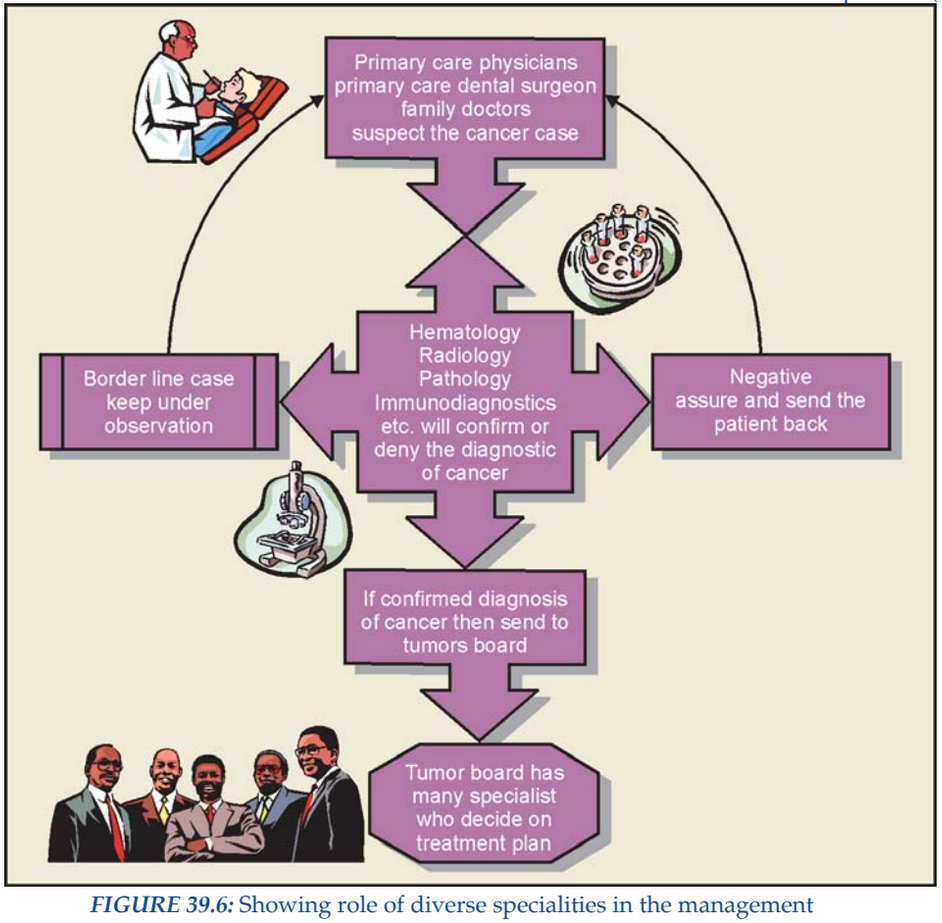


FIGURE 39.6: Showing role of diverse specialities in the management of oral cancer patient (Ramdas K, Bailoor DN 2004)

The Tumor Board rapidly reviews all the cases once the diagnostic procedures are complete. The Tumor Board decides about the policy of treatment after reviewing all the records (Figs 39.5 and 39.6).

Role of Radiotherapy

The use of radiation therapy in the management of squamous cell carcinoma of the head and neck is based on the following principles.[3]

1. Squamous cell carcinoma is usually radioresponsive and in early stage highly radiocurable.
2. The more differentiated the tumor the less rapid the radiation response and resolution and the higher the radiation dose required.
3. Exophytic and well oxygenated tumors are more radio-responsive than deeply ulcerative and infiltrative hypoxic tumors.
4. Squamous cell carcinoma, when limited to the mucosa are highly radio-curable.
5. Bone and muscle involvement by carcinoma adversely alters radioresponsiveness and subsequently decrease radiocurability.
6. The early small metastases can be controlled with radiation therapy alone. Advanced cervical metastatic lymph nodes are better treated with combined surgery and RT.

Indications of RT

1. T_1- T_2 lesions
2. T_3 T_4 locally-advanced lesions
   - Postsurgical treatment
   - Radiotherapy with or without chemotherapy especially for organ preservation.
3. Cervical lymph node
   - Elective treatment when no palpable lymph nodes present
   - Only treatment for clinically positive lymph nodes
   - Presurgical and postsurgical in combination with neck dissection for clinically positive lymph nodes.

Combined Surgery and Radiotherapy

Radiotherapy can be given before or after surgery. Each sequence has theoretical advantages and disadvantages. However, the results from randomized studies favor post-operative radiotherapy.[4,5] In practice, most surgeons also prefer to operate in an unirradiated field where frozen section control of resection margin can be obtained. In certain clinical settings, however, planned preoperative radiotherapy may be favoured. These include situations where cancer is doubtfully respectable or when a free osteomyocutaneous graft is to be used for mandibular soft tissue reconstruction. In the latter situation avoidance of irradiating the graft and delivery of a lower dose to the mandibular stump than would be necessary in post-operative setting both facilitate integration of the vascular graft.

Indications for Postoperative Radiotherapy

- Positive resected margins
- Multiple involved nodes
- Extracapsular extension
- Locally-advanced primary regardless of margin
- Perineural spread
- Vascular and lymphatic emboli

Timing of Radiation

The general guideline is to commence radiotherapy when tissues are well healed. The longer the interval before the commencement of radiation, the greater the opportunity for presumed clonogens to proliferate. A delay of more than 6 weeks can adversely affect the outcome.[6]

Combined Chemotherapy and Radiotherapy Treatment

Chemotherapy is generally used along with radiation in organ conservation settings, especially in locally advanced tumours to avoid surgery. Chemotherapy can be combined with radiotherapy in various ways.[7]

Neoadjuvant/Anterior/Induction Chemotherapy

Chemotherapy is given before local treatment like radio-therapy and surgery. This has 2 percent survival benefit.

Concurrent or Concomitant Chemotherapy

Chemotherapy is given along with RT. This has 8 percent survival benefit.

Adjuvant Chemotherapy

Chemotherapy is given after local form of treatment like radiotherapy or surgery. This has 1 percent survival benefit.

Preradiotherapy Evaluation

General

- Avoid endodontics
- Extract teeth with periapical lesions, carious exposures 2 weeks before RT.
- Antibiotic use dependent on patient and treatment modality.
- Fluoride (neutral sodium fluoride 1%) application tooth pastes, trays
- Avoid periodontal treatment; extract teeth with more than 4-6 mm pockets, grade 2 mobility and furcation involvements of grade 2 or greater.
- Remove partially-erupted third molars, may leave full bony impacted teeth if surgical difficulty, healing time, would delay radiation therapy significantly or if the lack of signs or symptoms indicates minimal future problems

Table 39.1: Dental surgeons are involved at all stages of treatment of oral cancers

Pretreatment Evaluation	*Posttreatment follow up*	*Long-term counseling/ diet/life style etc*
Suspect clinically	Maintain oral hygiene	Counsel regarding habits
Biopsy and hematolgy	Look out for recurrence	Diet of fresh vegetables and more fiber
OPG and other radiographs	Manage mucositis radiation caries, xerostomia etc.	Life style modification of lower stress/ Exercise/reduce spices/salt etc

Dose of Radiotherapy

The radiation dosage is determined by:

i. Tumor site
ii. Size of the lesion
iii. Volume to be irradiated (Target volume)
iv. No. of fractions of treatment.
v. Various techniques of delivery of Radiotherapy.
vi. Tolerance of various structures.
vii. Associated medical conditions like diabetes, collagen disorders etc

In general a doze of 50-55Gy in 25-30 fr. Over 5-6 weeks is considered, adequate for sterilization of microscopic or occult diseases.[8] A doze of 65-70 Gy over 7 weeks is required for control of gross tumor. The initial dose is usually 50Gy to the primary lesion and the regional nodes using a wide field technique followed by boost dose upto 65-70Gy using smaller volume covering gross disease. Spinal cord is shielded after 45Gy.

Post-treatment Protocol

- Care of teeth—fluoride application and regular check up
- Regular use of sodabicarbonate mouthwash
- Pilocarpine 5 mg tablet thrice daily to increase salivary flow
- Proper nutrition—especially proteinaceous food
- Post treatment follow up at regular intervals
- Regular exercise and physiotherapy
- Counseling and group therapy and other methods for proper emotional adaptation of the patient
- Avoidance of all surgical procedures involving bone for a minimum of six months to avoid osteoradionecrosis. Extraction of carious teeth can be done under antibiotic cover and the periosteum may be sutured if necessary.

Role of Oral Physician

Oral physicians should be a part of oral cancer care team and should contribute their expertise for the betterment of the patient. In the present scenario this aspect is not properly emphasized (Tables 39.1 and 39.2).

Table 39.2: Survival rates in oral cancer

Relative survival rates in 11375 cases

Site	*Stage*	*No of cases*	*5-year survival rate*
Lip	I-II	1125	95
	III-IV	288	78
Oral tongue	I-II	787	67
	III-IV	866	20
Floor of mouth	I-II	324	68
	III-IV	1669	41
Gingiva	I-II	70	55
	III-IV	822	44
Buccal mucosa	I-II	58	78
	III-IV	256	40

Follow-up Protocols

After completion of treatment patients will be asked to come for follow-up after 6 weeks and later at 3 months interval for the next two year and thereafter at increasing intervals.

Newer Radiation Delivery Techniques

New radiation delivery techniques offer powerful potential to diminish the severity of radiation toxicities. In conventional radiotherapy because of close proximity of head and neck tumors to critical organs like spinal cord, eyes, optic chiasma, etc. quite often delivery of maximal dose to tumor is not feasible. Advances in computer technology and engineering have facilitated delivery of higher dose to the tumor sparing critical organs.

1. **3D Conformal Radiotherapy**
   In this the treatment volume conforms to the shape of the tumor using 3D planning technique, minimizing the dose to normal structures.
2. **Intensity Modulated Radiotherapy (IMRT)**
   IMRT refers to a specific technique of linear accelerator based radiotherapy by which radiation beams are modulated in such a manner as to produce highly conformal dose distribution with in the tumor while reducing the dose to normal structures.

Drugs Used to Minimize the Side Effects of Radiotherapy

Amifostine: Amifostine formerly known as WR-2721 protects cell damage by scavenging free radicals. A dose of amifostine 200mg/m^2 is given as rapid infusion 30 minutes before each fraction of radiation. This can minimize the side effects of radiotherapy.

Pilocarpine: This drug is used against dryness of mouth to increase the salivary flow.

Erythropoietin: 10,000 units given as subcutaneous injection 3 times a week is used to correct anemia before radiotherapy.

Colony –stimulating factor (CSF): G-CSF and GM-CSF are used to diminish myelosuppression associated with chemoradiotherapy. These agents also provide mucosal protection in patients treated with chemoradiotherapy.

Palliative Radiotherapy

Locally-advanced head and neck cancer can benefit from palliative radiation inorder to control localized symptoms including respiratory compromise, bleeding, discharge from exophytic lesions, functional difficulties in speech and swallowing, cranial nerve involvement and avoid fungation of lymph nodes. These patients are treated with short course of radiotherapy, unlike more protracted course in a curative setting.

Radiotherapy—Indian Scenario

The history of radiotherapy services in India goes far back to 1910's in contemporary institutes in Patna, Madras and Lucknow[9,10] (Table 39.3).

- Barnard Institute of Radiology and Cancer, Madras
  The institute was opened on 17th March 1922 and a 200 KV deep X-ray unit was installed in 1924 and the first double coil 200KV unit installed in the East.
- Radium Institute, Patna Medical College Hospital, Patna

In 1913, Col Vougham of Indian Medical Service purchased Radium about 10 mgm and used them on cases of cancer at Ranchi. In July 1928, Radium Institute was shifted from Ranchi to Patna.

In the early 50's only deep X-ray units operating in the 200-300 kV range were available for teletherapy; the quality and the depth dose of these units were very poor. Of course the radium sources were used extensively for brachytherapy in most of the centers in India.

Table 39.3: List of old centers and year of establishment

Sl. no.	*Centers*	*Year*
1	Ranchi	1913
2	Lucknow	1918
3	Calcutta (MC)	1920
4	Indore	1920
5	Madras	1922
6	Patna	1928
7	Trivandrum	1938
8	Vellore	1939
9	TMH Bombay	1941
10	Agra	1944
11	Mysore	1947

First cobalt-60 teletherapy unit (Eldorado A)—1956 Cancer Institure, Chennai;

First linear accelerator - 1976 Cancer Institute, Chennai

Table 39.4: No of radiotherapy centers in India

Radiotherapy centers	166
Facilities for brachytherapy	110
Telecobalt machines	221
Linear accelerators	36
Cs^{137} units	10
Simulators	37
Treatment planning system	30
Nuclear medicine centers	140
Facilities for radioiodine treatment	25

As per an assessment by WHO and IAEA, developing countries like India need one radiation therapy machine per million of population. USA has 8.2 and UK has 3.4 such machines per million population. Accordingly, India needs at least 1000 such machines for cancer treatment. Thus there is a wide gap between the requirement and availability due to exorbitant cost of purchase (typically Rs 7-8 crore) and maintenance of imported machines. Unlike USA where 95 percent of radiotherapy is carried out with linear accelerators, in this country CO^{60} machines are in the mainstay for cancer treatment.

LIST OF REGIONAL CANCER CENTERS (Table 39.4)

1. Kidwai Memorial Institute of Oncology, Bangalore (Karnataka).
2. Gujarat Cancer and Research Institute, Ahmedabad (Gujarat).
3. Cancer Hospital Research Institute, Gwalior (Madhya Pradesh).
4. Cancer Institute, Madras (Tamil Nadu).
5. Regional Cancer Centre, Thiruvananthapuram (Kerala).
6. A H Regional Centre, Cuttack (Orissa).
7. Dr BB Cancer Institute, Guwahati (Assam).
8. Chittaranjan National Cancer Institute, Kolkata (West Bengal).
9. Institute Rotary Cancer Hospital (AIIMS), New Delhi.
10. Tata Memorial Hospital, Mumbai (Maharashtra).
11. Kamala Nehru Memorial Hospital, Allahabad (UP).
12. MNJ Institute of Oncology, Hyderabad (Andhra Pradesh).
13. RST Cancer Hospital, Nagpur (Maharashtra).
14. Indira Gandhi Institute of Medical Sciences, Patna (Bihar).
15. Acharya Harihar Tulsi Das Regional Cancer Centre, Bikaner, Rajasthan.
16. Indira Gandhi Medical College, Shimla (Himachal Pradesh).
17. Post-Graduate Institute of Medical Sciences, Rohtak (Haryana).
18. Pt. J.N.M. Medical College and Hospital, Raipur, Chattisgarh.
19. JIPMER, Pondicherry.

CONCLUSION

Radiotherapy is one the important options in the treatment of oral cancer. A dental surgeon needs to know the basics of radiation therapy because he forms a vital part of the team of health care professionals involved in managing this dreaded scrouge on humanity. His role in diagnosis, treatment and follow-up of radiotherapy patients is highlighted in this discussion.

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40 Complementary and Alternative Medicine: Emerging Vistas in Healing

INTRODUCTION

"Complementary alternative medicine is a group of diverse medical and health care systems, practices, and products that are NOT presently considered to be part of conventional medicine."

Many dentists work in conjunction with other alternative health care providers, such as homeopathic practitioners and holistic medical doctors. The dental procedures most commonly offered are neural therapy, ayurveda, oral acupuncture, cold laser therapy, and mouth balancing. To enhance the effectiveness of these therapies, the practitioner may prescribe homeopathic and ayurvedic remedies and make recommendations concerning diet and nutrition.

OTHER NAMES OF ALTERNATIVE MEDICINES

- Alternative medicine
- Complementary medicine
- Unconventional medicine
- Holistic medicine
- Natural medicine
- Integrative medicine

CLASSIFICATION OF ALTERNATIVE MEDICINE

Alternative medicine was classified into mainly seven groups. They are as follows;

I. **Alternative system of medical practice:**
   a. Ayurveda
   b. Homeopathic medicine
   c. Acupuncture
   d. Traditional Chinese medicine
   e. Tibetan medicine

II. **Herbal medicine**: Employing plants and plant products from folk medicine traditions for pharmacological use.
   a. Ginger rhizome
   b. *Ginkgo biloba* extract
   c. Ginseng root
   d. Echinacea

III. **Bioelectromagnetic application:** Study of how living organisms interact with electromagnetic field.
   a. Blue light treatment
   b. Electro-acupuncture
   c. Electro-stimulation
   d. Magnetoresonance spectroscopy

IV. **Mind/ Body control:** Exploring the minds capacity to affect The body, based on traditional medical systems that make use of the inter-connectedness of mind and body.
   a. Art therapy
   b. Counseling
   c. Humor therapy
   d. Hypnotherapy
   e. Dance therapy
   f. Meditation/Yoga
   g. Music therapy
   h. Quantum healing

V. **Diet/ Nutrition, lifestyle change:** Prevent illness/ maintain health; reverse the effect of chronic diseases through dietary or nutritional intervention.
   a. Diet and lifestyle changes
   b. Gerson therapy
   c. Macrobiotics/Megavitamins

VI. **Manual healing:** Using touch and manipulation with the hands as a diagnostic and therapeutic tool.
   a. Acupressure
   b. Trager method
   c. Zone therapy
   d. Massage therapy

VII. **Pharmacological biologic treatment:** Drugs and vitamins not yet accepted by mainstream medicine.
   a. Anti-oxidizing agent
   b. Cell treatment
   c. Chelation therapy
   d. Metabolic therapy

AYURVEDA

"Life (ayu) is the combination (samyoga) of body, senses, mind and reincarnating soul. Ayurveda is the most sacred science of life, beneficial to humans both in this world and the world beyond."

—Charaka Samhita, Sutrasthana

Ayurvedic medicine is a comprehensive multifaceted system of healing that originated in ancient India. In Sanskrit, ayur means life or living, and veda means knowledge, so Ayurveda has been defined as the "knowledge of living" or the "science of longevity." Ayurvedic medicine utilizes diet, detoxification and purification techniques, herbal and mineral remedies, yoga, breathing exercises, meditation, and massage therapy etc as holistic healing methods. Ayurvedic medicine is widely practiced in modern India and has been steadily gaining followers in the West.

According to the original texts, the goal of Ayurveda is prevention as well as promotion of the body's own capacity for maintenance and balance. Ayurvedic treatment is non-invasive and non-toxic, so it can be used safely as an alternative therapy or alongside conventional therapies. Ayurvedic physicians claim that their methods can also help stress-related, metabolic, and chronic conditions. Ayurveda has been used to treat acne, allergies, asthma, anxiety, arthritis, chronic fatigue syndrome, colds, colitis, constipation, depression, diabetes, flu, heart disease, hypertension, immune problems, inflammation, insomnia, nervous disorders, obesity, skin problems, and ulcers.

Ayurvedic physicians seek to discover the roots of a disease before it gets so advanced that more radical treatments are necessary. Thus, Ayurveda seems to be limited in treating severely advanced conditions, traumatic injuries, acute pain, and conditions and injuries requiring invasive surgery. Ayurvedic techniques have also been used alongside chemotherapy and surgery to assist patients in recovery and healing. Ayurvedic medicine originated in the early civilizations of India some 3,000-5,000 years ago. It is mentioned in the *Vedas*, the ancient religious and philosophical texts that are the oldest surviving literature in the world, which makes Ayurvedic medicine the oldest surviving healing system. According to the texts, Ayurveda was conceived by enlightened wise men through 10,572 hymns in Sanskrit as a system of living harmoniously and maintaining the body so that mental and spiritual awareness could be possible. Medical historians believe that Ayurvedic ideas were transported from ancient India to China and were instrumental in the development of Chinese medicine (Fig. 40.1).

In 1500 B C there were two ayurvedic schools. Atreya (school of physicians) and Dhanvanthari (school of surgeons). Charaka was famous physician and Sushrutha was surgeon. Charaka explained two main causes of any diseases;

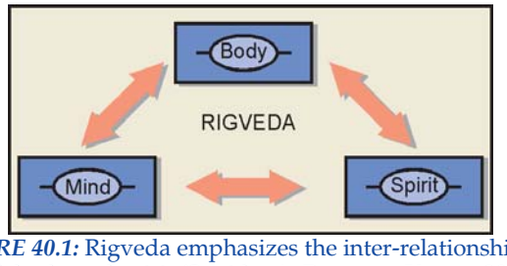

FIGURE 40.1: Rigveda emphasizes the inter-relationship of mind, body and spirit (Sunitha A, Prasanna Kumar 2004)

- ***Internal cause:*** Loss of faith in divine. This is beginning of spiritual, mental and physical disease.
- ***External cause:*** Time of day, season, diet and life style.

Sushrutha explained the surgical equipments, classification of abscesses, burns, fractures, wounds, amputation and plastic surgery/ rhinoplasty etc. In ayurvedic massage therapy they are mentioned about marma points or vital body points which is similar to Chinese acupuncture.

Over the centuries ayurveda has had a nurturing influence on ancient Chinese systems of medicine, Unani medicine, and the humoral medicine practiced by Hippocrates in Greece. The current knowledge about this ancient Indian medicine is primarily drawn from the *Charaka Samhita* (though there are earlier versions, *Charaka Samhita* in its present form is estimated to date from 1st century AD), Vagbhatta's *Astanga Hridaya* (approximately 500 AD), and the *Susruta Samhita* (the *Susruta Samhita* is believed to have originated in the last centuries BC, but the date of its present version is fixed by researchers at 7th century AD). These three classic texts describe the basic principles and theories from which this alternative medicine has evolved. They reflect an overwhelming wealth of clinical as well as surgical information, enriched further by later research, on the management of a multitude of diseases and ailments.[8]

Eight branches of ayurveda:

- Kayachikitsa (Internal Medicine)
- Shalakya tantra (ENT, Ophthalmology and Dentistry)
- Shalya tantra (Surgery)
- Agada tantra (Toxicology)
- Bhuta vidya (Psychiatry)
- Kaumarabhritya (Pediatrics)
- Rasayana (Anti-aging)
- Vajikarana (The science of fertility).

Saptha dathu theory:

Human body is composed of seven tissues called *dhatus*.

- Plasma and interstitial fluids (*rasa*)
- Blood (*rakta*)
- Muscle (*mamsa*)
- Fat or adipose tissue (*medas*)
- Bone (*asthi*)
- Bone marrow (*majja*)
- Reproductive tissue (*sukra*)
  - — Malas-Sweda/Mala/Muthra (Excretory products —helps in diagnosis)
  - — Srotas—opening and vessels (Srotarodha-vessel related diseases)
  - — Tripod—Doshas, Dhatu, Malas
  - — Agni and Aama—Digestion and Toxins
  - — Ojas—Immune system
  - — Prakruti—Mind and Body
  - — Gunas (mental status)
    - — Satva—Perfect
    - — Rajas—Semibalanced
    - — Tamas—Unbalanced

Stages of disease progression:

- Accumulation
- Aggravation
- Overflow
- Relocation
- Manifestation
- Diversification

PATHOPHYSIOLOGY OF DISEASES

Health has been defined in this Sanskrit verse "*Samadosha Samagnischa Samadhathumalakriyha Prasannathmendriya Manaha Swasthaithyabhidheeyate*"

According to Ayurveda health is defined as "*the state of body when balanced doshas, balanced digestion and metabolism, proportionate tissues (Dhatu), balanced function of all systems, cheerful disposition of senses, mind and spirit is achieved*."

There are five basic elements that contain prana: earth, water, fire, air, and ether. These elements interact and are further organized in the human body as three main categories or basic physiological principles in the body

that govern all bodily functions known as the *doshas*. The three doshas are *vata, pitta, and kapha*. Each person has a unique blend of the three doshas, known as the person's *prakriti*, which is why Ayurvedic treatment is always individualized. In Ayurveda, disease is viewed as a state of imbalance in one or more of a person's doshas, and an Ayurvedic physician strives to adjust and balance them, using a variety of techniques.[9]

The dosha concept is the most fundamental and characteristic principle of Ayurvedic healing. A comprehensive method to classify human types and diseases, treatment modalities and is pivotal to the entire system of Ayurvedic healing.

Vata: Normally comprehends all phenomenon under functions of central and sympathetic nervous systems. It sustains the body with expiration, inspiration, enthusiasm, and movement of various parts, keenness of sense perceptions, initiation of natural urges and other functions.

Pitta: Signifies functions of thermogenesis, or heat production and metabolism, the process of digestion, coloration of blood and formation of various secretions and excretions are either the means or ends of tissues combustion. Sustains body with digestion, heat production, desire, production of hunger, thirst, color and complexion, understanding, intelligence, valor, softness of body parts and similar others.

Kapha: Implies the functions of thermotaxis or heat regulation and secondarily formation of various preservative fluids (eg- Mucous synovia, etc). Sustain body with stability, lubrication, compactness of joints, visibility, forbearance, intelligence, courage, strength, attachment etc.

Diseases of the face and oral cavity are classified in 'Mukha Rogas'.

- *Sheetada/Scurvy gingivitis*—Gingiva is soft and spongy, bleeds on probing, painful.

Treatment: General principle

- Swedana (sweating therapy)
- Raktha mokshana (blood letting)
- Pratisarana yogas (preparations)
- Gandoosha or Kavalagraha (gargle, mouthwash)
- Nasya
- Internal yogas (medicine preparation)

Own Tongue as a Mirror Image of Body

An ancient art of tongue diagnosis also describes quite characteristic patterns, which can reveal the functional status of respective internal organs merely by observing the surface of the tongue (Fig. 40.2). A discoloration and/or sensitivity of a particular area of the tongue indicate a disorder in the organ corresponding to that area. A whitish tongue indicates a *kapha* derangement and mucus accumulation; a red or yellow-green tongue indicates a *pitta* derangement; and a black to brown coloration indicates a *vata* derangement. A dehydrated tongue is symptomatic of a decrease in the *rasa dhatu* (plasma), while a pale tongue indicates a decrease in the *rakta dhatu* (red blood cells).

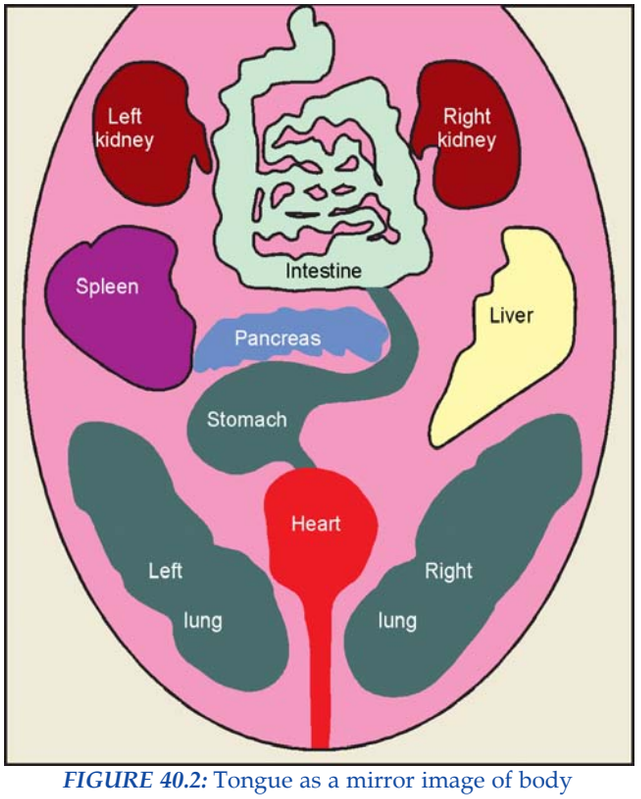

FIGURE 40.2: Tongue as a mirror image of body

Ayurveda insists that the 'fault' or *dosha*, the 'tissue' or *dhatu* and the 'impurity' or *mala* should be in harmony with each other, with all the components properly balanced. Any discordant note in this synthesis due to external or internal causes is a cause for concern. These are basically therapeutic measures taken either to prevent diseases or cure them. Thus ayurvedic procedures are done

either to detoxify the body or as a prelude to strengthening the immune system. *Panchakarma* or 'five procedures', is the most sought after detoxification therapy, which paves the way for the culmination of ayurvedic treatment of healing—anti-aging (*Kaya Kalpa*).

Three important factors in causing disease:-

1. **Prajnapradha—**Mistake of the intellect or wrong understanding of the environment can lead to disease. It is the main cause.
2. **Asatmyendriyartha samyoga—**Wrong association of sense objects with the sensory apparatus.
3. **Kala parinama—**i.e. the effect of time

Loss of equilibrium may be: -

- Dietary indiscrimination
- Undesirable habits
- Non-observance of rules of healthy living
- Seasonal abnormalities
- Improper exercise
- Erratic application of sense organs and incompatible actions of body and mind can also result in creating disturbance of the existing normal balance.

Ayurveda is a comprehensive health care system that can easily be integrated into current national healthcare and therapies. Most other alternative systems appear to have evolved from ayurveda. The potential benefits associated with the integration of ayurveda in to main stream medicine could include both patient satisfaction and a decrease in national healthcare cost, addressing demands not met by orthodoxy due to lack of consideration of a person as whole. As a science, ayurveda needs recognition, respectability and above all, reasonable regulation.

NATUROPATHY

Vincent Priessnitz (1799-1851)— "*Father of Naturopathy*"[10]

1. According to naturopathy all disease, their cause and their treatment are one.
2. The basic cause of disease is not bacteria. Bacteria develop after the accumulation of morbid matter when a favorable atmosphere for their growth develops in body. Basic cause is morbid matter and not the bacteria.
3. Acute diseases are our friends, not enemies. Chronic diseases are the outcome of wrong treatment and suppression of the acute diseases.
4. Nature is the greatest healer. Body has the capacity to prevent diseases and regain health.
5. Patient is treated and not the disease.
6. Patients suffering from chronic ailments are also treated successfully in comparatively less time.
7. Nature cure treats physical, mental, social (moral) and spiritual all four aspects at the same time.
8. This system treats body as a whole instead of giving treatment to each organ separately.
9. Naturopathy does not use medicines. According to Naturopathy, "Proper Food is the Best Medicine".

Nature cure includes all the available non-invasive treatments and diagnostic modalities, which do not interfere with the body's natural functional capacity and healing process and are in affirmation with nature's constructive Principles.

MUSIC THERAPY

Music is a universal language. It spans continents, languages and sensibilities. From time immemorial, music has been a part of Indian culture. All forms of celebrations have always been accompanied by music. The type of music varies according to religion, region and customs. But its appeal is universal. The saints and seers of ancient India recognized the importance of music. Music can play an effective role in helping us lead better, fruitful lives. Listening to specific kinds of music at specific times of the day has been shown to be helpful in maintaining good health. Indian music, with its many Ragas, is known to be particularly therapeutic.

In the Vedas too, music has an important place. The 'Sam Veda' is full of music. Hymns have been known to have a positive effect on human beings. The doshas like Vata, Pitta and Kapha can be controlled effectively by music therapy. Music can play an effective role in helping us lead better, fruitful lives. Listening to devotional music in the early hours of the morning gets one ready to face the challenges that the day has to offer. In the evenings, after a stressful day at work, the right kind of music helps one relax and refresh. Even during the course of working, light

music improves efficiency. Listening to music helps control negative aspects of our personalities like worry, bias and anger. In addition, it can help cure headache, abdominal pain and tension. Music therapy is one of the most effective ways of controlling emotions, blood pressure and restoring the functioning of the liver. The law of physiology states that when the mind is concentrated, the blood circulation is balanced. Such a body with a balanced circulation cannot be affected by any disease. Music can, therefore, play an important role in keeping us healthy.

ACUPUNCTURE

- It originated in India, after Budha's era it was transported to china and was developed there
- "Body has its own defense mechanism to control and cure the disease"
- It helps to controlling polio, asthma, arthritis, joint disorders, impotence, diabetes, headache etc.
- Acupressure gives slower effect in same conditions

Principle of Acupuncture

"There are certain points on the various parts of the body and on palms and soles which are related with 14 major meridans in the subtle body. Human potential energy has positive (+) and negative (-) factors that flow through these meridians which enables the body to function at all levels."

According to Chinese medicine theory " The human body consists of energy called Qi (chee), Qi follows specific pathways called meridians. When these pathways become upset, the two opposing forces of nature namely Yin and Yang, fall out of balance". Acupuncture needles realign the Yin and Yang.

Acupuncture use very fine sharp needles in to the body points (marma points) and creates balance in controlling disease. Acupuncture triggers the body to release natural chemicals, endorphins that reduce pain (Fig. 40.3).

TIBETAN MEDICINE

Tibetan medicine is a traditional system of medicine, which has been practiced for over 2500 years and is still practiced today although Tibetans are now in exile. Tibetan medicine is one of the five major sciences, and it is called *gSoba Rig-pa*, the science of healing. It uses different kinds of ingredients such as herbs, trees, rocks, resins, soils, precious metals, saps etc. However, 95 percent of Tibetan medicine is based on herbs, and precious metals are used for the seven kinds of precious pill known as *Rinchen rilpo*.

- All diseases categorized with in *"Nyipa sum"*, combination/balance of three poisons.

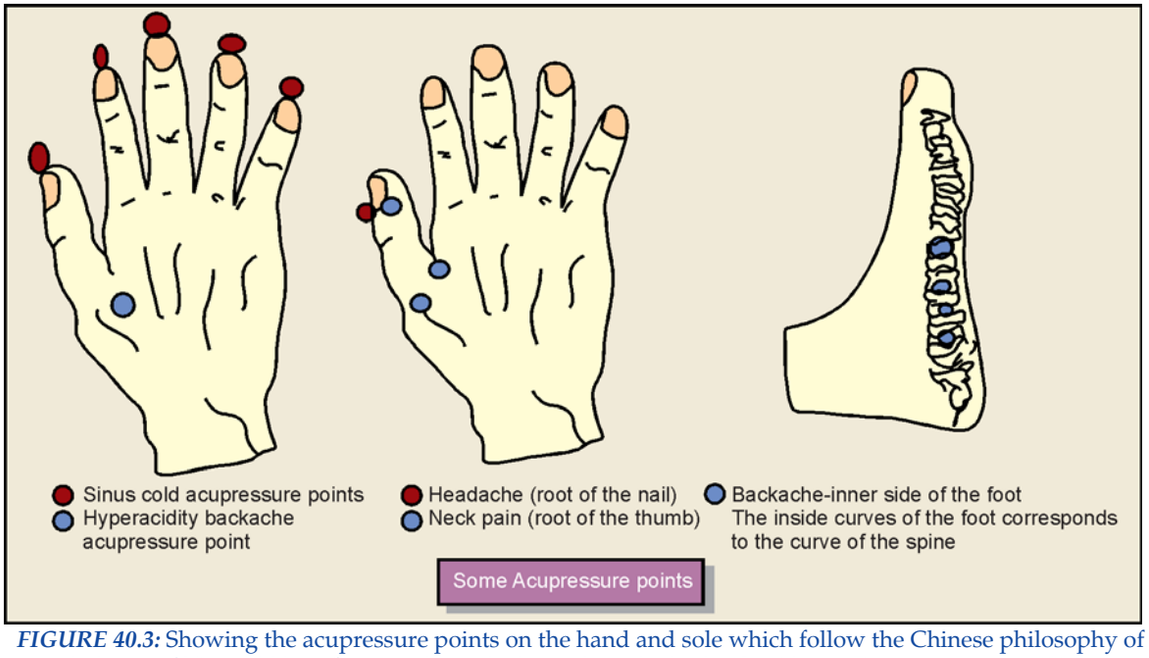

FIGURE 40.3: Showing the acupressure points on the hand and sole which follow the Chinese philosophy of balance of Yin and Yang flowing through channels in the body (Sunitha A, Prasanna Kumar 2004)

Theory of Tibetan Medicine

The basic theory of Tibetan medicine is to keep in balance the *Nyipa sum* - they are rLung (pronounced loong), *mKhris-pa* and *Bad-kan*. The long-term causative factors of *Nyipa sum* are the three poisons of desire, hatred and delusion which show how closely connected Tibetan medicine is with Buddhist philosophy.

- *rLung*—Flow of energy (help growth)
- *mKhris-pa*— Heat within the body
- *Bad-kan*— Sustain the bodily liquid

There are four tantras that explain the diseases in tibetan medicine. They are as follows:

- 1st Tantra—Explains the all diseases.
- 2nd Tantra—Explains the anatomy and physiology of body.
- 3rd Tantra—Explains the etiology, nature and disease classification.
- 4th Tantra—Explains the diagnosis and treatment.

HOMEOPATHY MEDICINE

"Aim to stimulate the body's defense mechanisms and healing processes by administering minute doses of plant extracts and minerals".

The word '*Homeopathy*' is derived from two Greek words, *Homois* meaning similar and *pathos* meaning suffering. Homeopathy simply means treating diseases with remedies, prescribed in minute doses, which are capable of producing symptoms similar to the disease when taken by healthy people. It is based on the natural law of healing- "Similia Similibus Curantur" which means "likes are cured by likes". The principle of Homeopathy has been known since the time of Hippocrates from Greece, the founder of medicine, around 450 BC. More than a thousand years later the Swiss alchemist Paracelsus employed the same system of healing based upon the principle that "like cures like." But it was not until the late 18th century that Homeopathy as it is practiced today was evolved by the great German physician, Dr. Samuel Hahnemann. He was appalled by the medical practices of that time and set about to develop a method of healing which would be safe, gentle, and effective. He believed that human beings have a capacity for healing themselves and that the symptoms of disease reflect the individuals struggle to overcome his illness.[6]

Homeopathy is the system of treatment based on demonstrable laws and principles, which are -

a. *The Law of Similars*: It is also called the Law of Cure. This law demonstrates that the selected remedy is able to produce a range of symptoms in a healthy person similar to that observed in the patient, thus leading to the principle of *Similia Similibus Curentur i.e. let likes be treated by likes*.

b. *The Law of Single Remedy*: This law directs to choose and administer such a single remedy, which is most similar to the symptom complex of the sick person at a time.

c. *The Law of Minimum Dose*: The similar remedy selected for a sick should be prescribed in minimum dose, so that when administered there is no toxic effects on the body. It just acts as a triggering and catalytic agent to stimulate and strengthen the existing defense mechanism of the body. It does not need to be repeated frequently.

HUMOR THERAPY

"The simple truth is that happy people generally don't get sick."

Humor is a universal language. It's a contagious emotion and a natural diversion. It brings other people in and breaks down barriers. Best of all it is free and has no known side reactions. Laughter is one of the easiest ways to free yourself from the mind's constant thought process and find inner peace. It will make people more alive, healthier, more creative, and more silent.

- Humor is a powerful antidote to stress
- It helps in muscle relaxation and
- Pain reduction
- An increase in the number and activity level of natural killer cells that attack viral infected cells and some types of cancer and tumor cells.
- An increase in activated T cells (T lymphocytes). There are many T cells that await activation. Laughter appears to tell the immune system to "turn it up a notch."
- An increase in the antibody IgA (immunoglobulin A), which fights upper respiratory tract insults and infections.

- An increase in gamma interferon, which tells various components of the immune system to "turn on."
- An increase in IgG, the immunoglobulin produced in the greatest quantity in body, as well as an increase in complement 3, which helps antibodies to pierce dysfunctional or infected cells. The increase in both substances was not only present while subjects watched a humor video; there also was a lingering effect that continued to show increased levels the next day.
- Women seem to benefit more than men in preventing hypertension.

Lee Berk and Stanley states that laughing lowers blood pressure, reduces stress hormones, increases muscle flexion, and boosts immune function by raising levels of infection-fighting T-cells, disease-fighting proteins called Gamma-interferon and B-cells, which produce disease-destroying antibodies. Laughter also triggers the release of endorphins, the body's natural painkillers, and produces a general sense of well being.

SPIRITUAL HEALING

Spirituality involves the recognition and acceptance of a God beyond our own intelligence and with whom we can have a relationship. This God can provide an experience of inspiration, joy, security, peace of mind, and guidance that goes beyond what is possible in the absence of the conviction that such a power exists. Spiritual healing is when energy is transmitted to the person who needs it. The treatment works on the body, mind and spirit, which are seen as one unit that must harmonize for good health. If a separate healer is involved, the healer will place the hand on the person being treated to channel the energy from the higher source. The spiritual healing can help mental and emotional problems and physical conditions such as a frozen shoulder.

The channeling of healing energy from its spiritual source to someone who needs it is called spiritual healing. The channel is usually a person, whom we call a healer, and the healing energy is usually transferred to the patient through the healer's hands. The healing does not come from the healer, but through him. On the other hand, you don't need a healer to take advantage of spiritual healing. The word "spiritual" refers to the divine nature of the energy, which healers agree comes from one external, invisible intelligent source. The healing energy from this source is available to all. Healers see the body mind and spirit as one interdependent unit and believe all three must work in harmony to maintain positive health. Any problem - be it a broken leg or depression needs the power of healing to restore the balance of the whole person. It is felt that sickness often starts in the mind, or at the deeper level of the spirit, and it is often here that healing begins. According to Gandhi ji praying according to one's spiritual faith is an important part of treatment.

MEDITATION

Meditation is one of the proven alternative therapies. It can be broadly classified under the mind-body medicine. More and more doctors are prescribing meditation as a way to lower blood pressure, improve exercise performance in people with angina, help people with asthma breathe easier, relieve insomnia and generally relieve the everyday stresses of life. Meditation is a safe and simple way to balance a person's physical, emotional, and mental states. It is simple; but can benefit everybody.

The use of meditation for healing is not new. Meditative techniques are the product of diverse cultures and peoples around the world. It has been rooted in the traditions of the world's great religions. In fact, practically all religious groups practice meditation in one form or another. The value of meditation to alleviate suffering and promote healing has been known and practiced for thousands of years.

Meditation reduces activity in the nervous system. The parasympathetic branch of the autonomic or involuntary nervous system predominates. This is the branch responsible for calming us. During anxiety and tension states there is a rise in the level of lactate in the blood. Lactate is a substance produced by metabolism in the skeletal muscles. During meditation blood lactate levels decrease at a rate four times faster than the rate of decrease in non-meditators resting lying on their backs or in the meditators themselves in pre-meditation resting.

Three Stages of Meditation

- *Dharana*—Concentration
- *Dhyana*—Contemplation
- *Samadi*—Control vital functions

Types of Meditation

There are various types of meditation— prayer is probably the best known, but there is also TM (Transcendental Meditation), mindfulness meditation, and from the Eastern tradition, Zen meditation, Buddhist meditation, and Taoist meditation.

- **Concentrative meditation:** Focuses the attention on the breath, an image, or a sound (mantra), in order to still the mind and allow a greater awareness and clarity to emerge. This is like a zoom lens in a camera; we narrow our focus to a selected field.
- **Mindfulness meditation**: It involves opening the attention to become aware of the continuously passing parade of sensations and feelings, images, thoughts, sounds, smells, and so forth without becoming involved in thinking about them. The person sits quietly and simply witnesses whatever goes through the mind, not reacting or becoming involved with thoughts, memories, worries, or images. This helps to gain a more calm, clear, and non-reactive state of mind. Mindfulness meditation can be likened to a wide-angle lens.

YOGA

Yoga's primary emphasis is upon general well being. Although yoga has been shown to be beneficial in a variety of conditions, it is not considered a therapy for specific illnesses. Rather, yoga employs a broad holistic approach that focuses on teaching people a new lifestyle, way of thinking, and way of being in the world. In the process, however, it is also found to bring a myriad of healing effects. By attending to practices for improving, regaining or retaining general good health, a person is likely to find that some of his more specific difficulties tend to disappear. Many of the healing effects of yoga are clinically verified. However, one of the most important benefits of yoga is its application in relieving stress and fatigue producing invigoration and vitality and its anti-aging properties and its application for relaxation therapy.

Yoga has been used for disorders such as: Acid stomach, Addictions, Asthma, Backache, Bronchitis, Constipation, Depression, Diabetes, Headache, Heart disorders, Hypertension (High blood pressure), Stress and Tension etc.

UNANI *(Based on the Greece Philosophy)*

Unani medicine is based on the Greece philosophy. According to Basic Principals of Unani the body is made up of the four basic elements, i.e. earth, air, water, fire which have different Temperaments i.e. cold, hot, wet, dry. After mixing and interaction of four elements a new compound having new temperament comes into existence, i.e. hot wet, hot dry, cold wet, cold dry. The body has the simple and compound organs, which got their nourishment through four, humors i.e. blood, phlegm, yellow bile, black bile. The humor also assigned temperament as blood is hot and wet, phlegm is cold and hot, yellow bile is hot and dry and black bile is cold and dry.

Health is a state of body in which there is equilibrium in the humors and functions of the body are normal in accordance to its own temperament and the environment. When the equilibrium of the humors is disturbed and functions of the body are abnormal, in accordance to its own temperament and environment, that state is called Disease. Unani medicine believes in promotion of health, prevention of diseases and cure. Health of human is based on the six essentials (Asbabe Sitta Zaroorya) if these are followed health is maintained otherwise there will be diseases.[10] Six essentials are:

- Atmospheric air
- Drinks and food
- Sleep and wakefulness
- Excretion and retention
- Physical activity and rest
- Mental activity and rest.

Diagnosis

Diseases are mainly diagnosed with the help of pulse (*Nabz*), physical examination of the urine and stool. Also, patients are examined systematically to make the diagnosis easy as spot diagnosis with the help of simple, modern gadgets.

Treatment

Diseases are treated in the following ways:

1. Ilajbil Tadbeer (Regimenal Therapy)
2. Ilajbil Ghiza (Dietotherapy)
3. Ilajbil Dava (Pharmacotherapy)
4. Ilajbil Yad (Surgery)

1. *Ilajbil Tadbeer(Regimenal Therapy):* Some drugless regimens are advised for the treatment of certain ailments i.e. exercise, massage, hamam(Turkish Bath), Douches(Cold and Hot) and the Regimen for Geriatrics.
2. *Ilajbil Ghiza(Dietotherapy):* Different diets are recommended for the patients of different diseases.
3. *Ilajbil Dava (Pharmacotherapy):* The basic concept of treatment is to correct the cause of the disease that may be Abnormal temperament due to
   I. Environmental factors
   II. Abnormal humors either due to internal causes or external causes which may be pathogenic micro-organism, through (a) drugs of opposite temperament to the temperament of the disease that is called Ilaj-bil-zid or (b) drugs of similar temperament as of the temperament of the disease that is called as Ilaj-bil-misl

The drugs used are mostly of the plant origin. Some drugs of animal and mineral origin are also used. Patients are treated either by single drugs (crude drugs) or by compound drugs (formulations of single drugs). There are two types of compound drugs used in the treatment of the diseases i.e. classical compound drugs which are in use for the hundreds and thousands years and Patent/Proprietary compound drugs which have been formulated by the individuals or institutions as per their research and experiences.

COMPLEMENTARY AND ALTERNATIVE MEDICINE IN DENTISTRY

- Krauss and colleagues mentioned that complimentary alternative medicine (CAM) was mainly used for chronic pain in western countries.
- It was also effective in temporomandibular disorders and chronic facial pain.
- Massage therapy, acupuncture, naturopathy, herbal medicines are the main CAM used.
- Pain was the most frequently cited reason for CAM.
- Acupuncture proved effective in 73 percent of cases.[1]
- It was also effective in dental pain, facial pain, and TMDs.
- Single use needle should be used.
- Acupressure and massage therapy was used for treating bells palsy.[2]
- It is also used in salivary diseases and Sjögren's syndrome and dentine hypersensitivity.[3-5]
- Homeopathy medicines reduce swelling after extraction, gum disease/abscesses/anxiety (Table 40.1).
- Homeopathic toothpastes are also available in the market.
- It has got fewer side effects.
- Meditation, yoga and music therapies has got important role in reducing hypertension and stress.
- Research into the use of acupuncture in dentistry is encouraged
- Proper training is necessary.

Table 40.1: Homeopathy drugs for dentistry[7]

Symptom	*Remedies that could be used to treat*
Toothache	Plantago, Coffea, Chamomilla, Mercurius, Staphisagria, Pulsatilla, Arnica, Belladona, Kreosotum, Apis, Bryonia
Gingivitis	Nitric ac, Mercurius corr.
Fear of treatment	Aconite, Gelsemium, Chamomilla
Tooth decay	Mercurius, Silicea, Kreosotum, Sulpher
Discomfort after treatment	Arnica, Hypericum, Ledum, Phosphorus, Ruta
Abscess	Mercurius, Belladona, Silicea

CONCLUSION

Significant gaps in the available scientific knowledge base, limit the ability of dental professionals to guide patients with regard to Complementary Alternative Medicine approaches to dental treatment. WHO has identified more than 300 alternative therapies. Together with modern system of medicine alternative health sciences/therapies offer cheaper and easily adaptable solutions to various ailments for positive health. The Dental surgeons should not try these treatments on their own but may collaborate with different specialists and see how each of these areas

can find their place in contemporary scientific dental medicine regimen.

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This chapter is for information only. The Editors do not endorse any treatments mentioned to be used by dental surgeons in their practice as Each of these specialties need to be practiced by doctor duly certified by government by the respective alternative medicine boards and as per the law existing in each state of India.

—Editors

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